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Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

	Page.
ORIGINAL ARTICLES—	
Sporadic Occurrence of Influenza in Victoria during 1943, by W. I. B. Beveridge and S. E. Williams	77
The Modern Management of Prostatic Obstruction, by R. J. Silverton	80
The Precipitin, Agglutination, Indole and Methyl Red Reactions of the Dysentery Bacilli, by F. Draper, B.Sc.	84
REPORTS OF CASES—	
Nephrotic Type of Nephritis in Association with Quartan Malaria, by K. S. Harrison and W. P. H. Dakin	90
LEADING ARTICLES—	
Pertussis: Some Aspects of its Prevention and Diagnosis	91
CURRENT COMMENT—	
Fostering Food Fads	92
Immunization in the United States Army	93
Hæmorrhage in Severe Liver Disease	93
ABSTRACTS FROM MEDICAL LITERATURE—	
Dermatology	94
Urology	95
BRITISH MEDICAL ASSOCIATION NEWS—	
Scientific	96
POST-GRADUATE WORK—	
Week-End Course at Lismore, New South Wales	98
CORRESPONDENCE—	
An Appeal	99
The Nervous Disorders of Service Personnel: An Urgent Problem	99
An Acknowledgement	99
A Severe Serum Reaction	99
AUSTRALIAN MEDICAL BOARD PROCEEDINGS—	
New South Wales	100
Tasmania	100
OBITUARY—	
William Algernon Andrews	100
Felix Paul Bartlett	100
Archie Sheridan Coburn	100
NOMINATIONS AND ELECTIONS	100
MEDICAL APPOINTMENTS	100
DIARY FOR THE MONTH	100
MEDICAL APPOINTMENTS: IMPORTANT NOTICE	100
EDITORIAL NOTICES	100

SPORADIC OCCURRENCE OF INFLUENZA IN VICTORIA DURING 1943.

By W. I. B. BEVERIDGE AND S. E. WILLIAMS,

From the Walter and Eliza Hall Institute of Research in Pathology and Medicine, and the Commonwealth Serum Laboratories, Melbourne.

DURING 1943 an investigation on upper respiratory infections was carried out at the Hall Institute in collaboration with the Australian army in Victoria. Two papers on the isolation of influenza A and B viruses have already been published.[1][2] This paper reports the epidemiological aspects of the investigation.

Every week the army authorities informed us of the number of troops "reporting sick" with upper respiratory tract infections in each military camp. These figures, accumulated for the whole of the military personnel in Victoria and worked out on the basis of a weekly incidence per thousand, have been used to prepare the graph in Figure I. The total number under observation cannot be disclosed beyond the indication that tens of thousands were involved.

From May until November regular visits were made to a camp hospital twice a week, and other camp hospitals were visited occasionally, especially whenever any appreciable increase in numbers of respiratory infections was reported there. On these visits patients were selected for study and specimens of blood and sometimes throat washings were obtained. Second specimens of blood were taken on the twelfth day after onset or later. Although as far as possible patients with an influenza-like syndrome were chosen, many were included whose illness did not conform to the classical picture. All patients were febrile at some stage.

The camp hospital that was regularly visited received patients from several permanently stationed units which had a considerable amount of contact with the civilian community of Melbourne, and also from a fairly large number of temporarily resident, itinerant military personnel, so that it probably provided a representative sample of infections present in the military and civilian communities.

Serological Investigation.

Each pair of sera, after being heated to 56° C. for thirty minutes, was titrated by the hæmagglutinin inhibition method of Hirst, with the modification adopted in this laboratory.[3] All sera were tested in this way for antibody rise against a "Melbourne" subtype of influenza virus A, strain BEL, and influenza virus B, strain BON.[3] Some were also titrated against the influenza virus B, strain LEE, the results being essentially the same as those obtained with BON.

A twofold or greater increase in titre of the second serum as compared with the first was considered diagnostic of infection having occurred with the type of virus concerned. In all, 98 pairs of sera were tested and eight infections with influenza A and 17 with influenza B were detected. No instance occurred of an increase in titre against both viruses. Those pairs of sera which did not show a twofold or more increase in titre gave the same titre with both specimens in nearly all instances. The details of the sera giving positive results are shown in Table I.

The antibody rises in cases of both A and B influenza in 1943 were generally somewhat smaller in degree than were the rises observed during the epidemic of influenza A in 1942. In 1943, in only one of the eight cases of influenza A and in three of the 17 cases of influenza B did eightfold or greater increases occur, whereas in 1942, in half of the 28 cases studied increases of this order occurred.

To confirm the hæmagglutinin inhibition titrations we selected at random four pairs of sera from the influenza A group and seven from the influenza B group and titrated them by the allantoic method.[4] The results are shown in Table I. The A strain BEL was used for the A cases, BON for the first three B cases and LEE for the last four. Nine of the 11 tests revealed a pronounced rise in antibody by this technique. In two of the influenza B cases, in which a good antibody rise to BON occurred, no rise was detected allantoically against the A strain BEL.

From the isolated case of influenza A which was detected in September, BEE, the following titrations were obtained with different strains: W.S. 15 to 30 (twofold), FOL 15 to 200 (thirteenfold), "Melbourne" 30 to 180 (sixfold), BEL 12 to 80 (sevenfold), BON 160 to 160. Thus the greatest increase (thirteenfold) was shown with the A strain FOL, which had been isolated three months previously from the camp in which this

TABLE I.

Serological Data of those Patients in whom Antibody Increases to Influenza Virus were Found.[1]

Subject.	Inhibition of Hæmagglutination.		Allantoic Titration.	
	First Serum.	Second Serum.	First Serum.	Second Serum.
		Influenza A.		
GRE[2] ..	15	80	<2	>7
COL[2] ..	10	40	<2	7
SPE ..	40	160	3	>7
MAR ..	10	30		
BAR ..	25	80		
FOL[2] ..	<10	160		
CLA ..	9	28		
BEE ..	12	80	<2	<2
		Influenza B.		
KEL ..	20	100	<2	6
BON[2] ..	20	60	3	7
KAN ..	20	80		
STE ..	30	60		
WHE ..	20	100	3	6
EDW ..	12	120		
McP ..	20	40	<1	<1
CLA ..	40	160		
BLU ..	<10	40		
BAT ..	18	80	<1	5
TEE ..	10	40		
FIT ..	15	60		
DUN ..	10	90		
SCH ..	15	55	<1	6
CLA ..	10	120		
SHE ..	20	70	<1	6
TIL ..	<10	30		

[1] In each of the two groups the subjects' names are arranged in the chronological order in which the cases occurred. Allantoic titres are expressed as the $\log_{10}$ of the number of infective doses neutralized by undiluted serum.

[2] Virus was isolated from these cases.

case BEE occurred. In the BEE sera no increase was detected against BEL on allantoic titration. However, this method of titration is highly specific, and the failure to show an increase may have been due to the fact that the strain causing the infection was not sufficiently closely related to BEL. There was a decided rise in antibody against "Melbourne", as shown by the neutralization test in mice. The first serum neutralized 1,000 lethal doses and the second 10,000.

Virus was isolated from throat washings from four of the influenza A patients and from one of the influenza B patients. This aspect of the investigation has been reported elsewhere.[1]

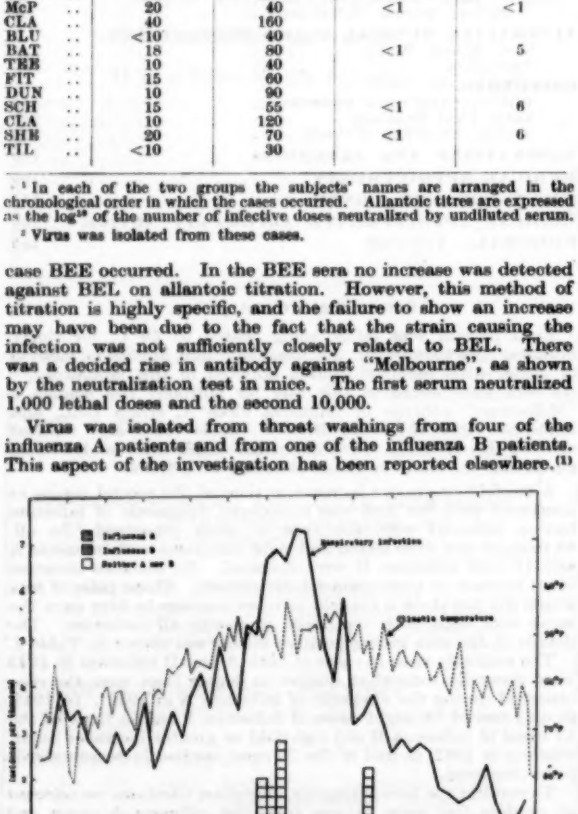

FIGURE I.

Graph showing the weekly incidence of upper respiratory tract infections in army personnel in Victoria in 1943, together with the half-weekly mean climatic temperatures at Melbourne and the results of our serological investigations. The temperature is plotted on an inverted scale. Each block represents one case investigated serologically.

Clinical Observations.

The patients who subsequently proved to be suffering from influenza B showed a mild, generalized illness, with some degree of localization to the upper respiratory tract. On admission to hospital these patients mostly complained of general weakness, malaise and anorexia. The temperature was usually 101° to 102° F., but never higher, and the elevation lasted from one to three days. About half of the patients complained of cough and a third of them had a sore throat. Headache was present in most cases. Pains in the limbs and back were frequently complained of, but they disappeared soon after admission to hospital. In the early stages the face appeared flushed and the eyes suffused. The pharynx was moderately congested in about half the cases and in two cases was severely inflamed and swollen. No signs worth noting were observed in the chest. The course of the illness was uneventful and recovery was uniformly complete, the majority of patients being returned to their units four to seven days after admission to hospital.

In many of the cases of influenza B the onset was insidious; the first sign was often a cold, which gradually developed into more general symptoms of malaise, limb pains, headache and feverishness. Some patients had symptoms for several days before reporting for medical examination. In only a few cases was there the sudden onset met with in most cases of influenza during epidemics.

When patients were examined in the wards, it was quite impossible to distinguish those who subsequently proved to be suffering from influenza B from the general group of those with upper respiratory infections.

The influenza A infections were on the whole somewhat more severe and had a more acute onset.

Epidemiological Data.

The graph in Figure I shows the weekly incidence of upper respiratory tract infections in military personnel in Victoria and its relationship to half-weekly mean climatic temperatures at Melbourne and the occurrence of influenza. It should be noted that this graph was compiled from the numbers of persons reporting for medical attention, who of course would be a small proportion of the number with minor upper respiratory infections. Between 25% and 40% of those reporting each week were admitted to hospital. Although there was a somewhat increased incidence of respiratory infections during June, July and August, at no time did the weekly incidence exceed 7·3 per 1,000.

Influenza A was present in May and June, and a single case was also detected in September. Influenza B occurred quite sporadically during most of the period of the investigation, cases being detected nearly every week from the middle of June till the end of September.

Both influenza A and influenza B occurred in widely separated localities, each disease being found in four different camps.

As the graph shows, there was no general epidemic of respiratory infections associated with the influenza cases which we detected. There were only three cases of influenza B out of 33 cases investigated (9%) during July, when there was a slight general increase in incidence of upper respiratory infections and of clinically diagnosed influenza, whereas there were 12 cases of influenza B out of 32 studied (38%) during August and September, when the general incidence had subsided.

Two minor prevalences of respiratory infections occurred in separate camps at different times. In one instance nine cases were studied, and provided one case of influenza A and one of influenza B. On the other occasion, when a unit recently arrived from the tropics was involved, only one case was studied, and the infection proved to be influenza A. All the other cases of influenza were quite unassociated with any local prevalence beyond the usual number of respiratory infections occurring at that time of the year. This statement is based on the weekly returns from the camps concerned and from personal consultations with the medical officers on the spot. It is doubtful whether the two minor prevalences were due primarily to influenza A, because at that time there was about the same proportion of cases of influenza A in camps where there was no prevalence.

The population from which the data for the graph were obtained was not fixed, a small proportion from time to time leaving for other areas and being replaced by new arrivals. Some units were brought here after a year or more of residence in the tropics, and it was observed on several occasions that these troops suffered a higher incidence of respiratory infections than did troops who had been in Victoria for some time. Such incidents may account for some of the small week-to-week variations in incidence shown in the graph.

In an attempt to obtain some information on the prevalence of influenza B in the community during the winter, titrations were carried out on sera from 30 medical students. Blood was

taken from the students in the autumn and early winter (March to June) and again at the end of September. None of them showed an increase in haemagglutinin inhibition titre against strain BON. None of these students reported having suffered from clinical influenza, but nearly all had had one or more afebrile colds.

The Epidemiology of Influenza B.

Influenza B is believed to have been the cause of at least two extensive epidemics with a high incidence as well as of several smaller outbreaks. In 1936 one occurred on the Pacific Coast of the United States of America, and in 1940 one involved several of the south-eastern States. Sera which had been kept from cases in these epidemics were subsequently tested against influenza B, and most tests showed that a rise in antibody against this virus had occurred.[5] Extensive serological studies carried out by Lennette and co-workers during 1940 and 1941[6] showed that both influenza A and influenza B were involved simultaneously in several outbreaks, some localized, some fairly extensive. However, there was a considerable predominance of one or the other at any one time or locality. In England there was a minor prevalence of influenza early in 1939; some of these infections were subsequently found to have been due to influenza B and others to influenza A.[7]

During 1941 in the Argentine, Taylor and his co-workers[9] investigated cases of *grippe* in a naval school. There was no clearly defined epidemic, but cases of influenza B were diagnosed serologically right through from June to October. This observation, coupled with the fact that a small proportion of influenza B cases had been found in several epidemics predominantly of influenza A, suggested that influenza B may commonly occur sporadically. Those epidemics studied by Lennette and co-workers,[6] which consisted mostly of cases of influenza A, but included some influenza B infections and were regarded by them as mixed epidemics, were probably essentially influenza A epidemics; but the large series of serological tests detected some influenza B cases which were occurring sporadically.

In the light of the further evidence provided in the present paper, it seems justifiable to regard influenza B as a disease which ordinarily occurs sporadically, but which at times gives rise to epidemics, either localized or widespread.

In a group of 31 children, aged from two to twelve years, on whom a serological study was carried out by Beveridge and Burnet,[10] there were only 12 who showed evidence of having had influenza B compared with 25 who showed evidence of having had influenza A. None of the 30 medical students studied showed evidence of having had influenza B during the winter of 1943. In the light of these two findings we cannot regard influenza B as being an especially prevalent disease—at least, not during recent years in Victoria. However, all of 55 adults whose serum was examined by Burnet[11] showed evidence of past infection, and our general experience with medical student volunteers has been that nearly all have been infected.

Discussion.

The epidemiology of influenza B has been fully discussed in the preceding section.

The diagnosis of influenza A has frequently been associated with widespread epidemics, but this has by no means always been the case. On several occasions[6][8][12][13][14][15] it has been found in localized epidemics, apparently confined to districts or institutions, and the following reports describe cases which probably occurred sporadically. In New York, in 1940, localized epidemics occurred, which Lennette *et alii* found were due mainly to influenza B (13 out of 24 cases); but three out of 24 cases were of influenza A. Similarly in Canada, in February, 1943, four cases of influenza A were found in 67 cases investigated, of which 32 were of influenza B.[8] During the winter of 1940-1941,[13] eight cases of influenza A were detected which occurred sporadically in London, no epidemic having been recognized there. In the light of the further sporadic cases detected by us in Victoria, in 1943, we are led to suggest that there has been also too great a tendency to regard influenza A as a disease found exclusively during epidemics.

The antibody rises recorded in the sporadic cases during 1943 were generally less than those found during the epidemic of influenza A in 1942. This may have been merely because in 1943 the sera were tested against strains which were less closely related to the strains causing the infections than was the case in 1942. The titrations of BEE sera with several strains give some support to this suggestion. Alternatively, it may be that strains causing an epidemic are more virulent and for this reason give rise to a greater antibody response.

The graph provides a useful illustration of the seasonal incidence of upper respiratory infections which is regarded here as normal. There was a broad general inverse correlation between the climatic temperature and the incidence of respiratory infections throughout the year, but the relationship was less close after August than before. This decrease in the apparent effect of temperature on the incidence of infections in late winter and spring may be due to acclimatization to colder temperatures or to naturally acquired immunity. A more detailed examination of the graph reveals several occasions (January 22, March 26, May 21), when a drop in the temperature was associated with a rise in the number of respiratory infections. However, temporary variations in the half-weekly average temperatures generally do not seem to have had a great influence on the incidence of infections. Infections increased in a fairly steady manner in June and July despite temperature fluctuations. This steady rise in incidence is suggestive of the cumulative effect of a spreading infective agent or agents—that is, the building up of a low-grade epidemic. At this time there was also an increase in the proportion of cases diagnosed clinically as influenza, but the proportion diagnosed serologically as influenza A or B was at its lowest during July.

The main clinical feature of the influenza B cases which calls for comment is that slow onset was more frequent than rapid onset, and that many of the infections commenced as a common cold which over several days gradually became more serious. Cases occurred without the slightest clinical indication that they differed ætiologically from the common respiratory infections always found in the winter. Most authors reporting clinical observations on influenza B describe a rapid onset as usual; but during 1943 Hare and his co-workers[8] found, as we did, that most of these infections had an insidious onset. These workers also detected some cases unassociated with fever.

Summary.

The weekly incidence of upper respiratory tract infections in military personnel in Victoria during 1943 was inversely correlated to the climatic temperature. This apparent influence of temperature was more pronounced before August than after. There was no clearly defined epidemic.

Influenza B occurred sporadically from June to September inclusive, and influenza A in May, June and September. Most cases were quite unassociated with any local or general prevalence of respiratory infections.

It is considered that influenza B usually occurs sporadically, although it sometimes takes on epidemic form. Sporadic cases of influenza B often have an insidious onset and cannot be diagnosed clinically.

Influenza A, although it usually occurs in epidemic form, does occasionally occur sporadically.

Acknowledgements.

We wish to thank Major-General S. R. Burston, Director-General of Medical Services, for permission to conduct this investigation, and Colonel Gordon Shaw, Deputy-Director of Medical Services, Victoria Lines of Communication Area, for his cooperation and help. We are indebted to the Royal Australian Air Force Meteorological Services for data on temperature. Our thanks are also due to Dr. F. M. Burnet for helpful advice and to Miss D. R. Bull for carrying out the allantoic titrations.

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THE MODERN MANAGEMENT OF PROSTATIC OBSTRUCTION.

By R. J. SILVERTON,
Sydney.

Two elements of the title of this article require comment. The word "modern" connotes a new outlook or a new method, recently introduced, and that is certainly so in this case. Bladder-neck obstructions are unfortunately common after middle age, and their treatment has been beset with manifold difficulties and dangers. During the past decade, however, a revolutionary change in the treatment of these obstructions generally has occurred, and during the past year an even more astounding transformation has been effected in the treatment of carcinomatous obstructions in particular. In 1926, Maximilian Stern, of New York, introduced the method of removing prostatic obstructions, through the urethra, by means of an endoscope furnished with a movable wire-loop electrode, activated by a high-frequency cutting current.(1) Stern predicted a glorious future for the new method, even though he reported on only 46 cases. He also predicted that the establishment of a permanent cystostomy for prostatic carcinoma would largely be replaced by endoscopic resection, and this, as well as his general prediction, has come true. In 1931, Joseph McCarthy, of New York, introduced his prostatic resectoscope, a more practical instrument than Stern's, with splendid visual control by means of his quite renowned "foroblique" telescope.(2) The latter is given this name because the vision is mainly forwards, with a slight oblique inclination towards the side aimed at. The same telescope is used with the McCarthy panendoscope, which is undoubtedly the most useful diagnostic endoscope available at the present time. McCarthy's resectoscope is so simply, strongly and efficiently designed that it has needed no essential modification since its introduction, and about three-quarters of American urologists performing endoscopic resection today prefer it to the punch and other types of instrument available. I shall discuss the importance and significance of perurethral resection later in this paper, but before leaving this introductory section I should like to refer briefly to the antiandrogen treatment of prostatic carcinoma, which is the second and more astounding element of the modernism referred to in my title. In 1941, Charles Huggins and co-workers at the University of Chicago noted extraordinarily beneficial effects in patients suffering from advanced carcinoma of the prostate simply from the performance of castration and nothing else. Apparently androgens activate prostatic epithelium, and their removal, or their neutralization by œstrogens, deactivates it. With this discovery were bound up observations on the presence of an enzyme, acid phosphatase, in normal prostatic tissue as well as in all kinds of carcinomatous tissue arising in this organ, and also in metastases from such carcinomata. Serum acid phosphatase increases in amount when metastases occur—that is, when much prostatic tissue is spread throughout the body—for, as it is concluded, prostatic carcinoma must partake of the nature of an overgrowth of adult epithelial cells rather than of neoplastic cells of a more primitive type. In any case, the simple removal or neutralization of androgens has lit with hope a very dark portion of the urological scene, and it may prove shortly that, even in the earliest cases of prostatic carcinoma, antiandrogen therapy is more effective than the difficult procedure of radical perineal prostato-vesiculectomy. Certainly in all advanced cases it has rapidly established itself as the treatment of choice, a small endoscopic resection being made to open the bladder outlet quickly and to yield tissue for biopsy.

The other element in the title requiring discussion is the word "prostatic". We have just seen that in many cases prostatic carcinoma is regarded as an overgrowth of adult prostatic epithelium. It has long been considered by many that prostatic carcinoma arises most often, if not always, in what is called the "true prostate", and not in a prostatic adenoma. The latter is an innocent neoplasm, which is often not really an adenoma and probably never truly prostatic. Clyde Deming, of Yale, recently described two phases in the development of the so-called "prostatic hypertrophy"(3); the first was an aglandular phase, really fibro-myomatous, exactly analogous to the uterine fibro-myoma, and arising in Müllerian muscle fibres in the embryologically female portion of the floor of the posterior part of the urethra. The accompanying diagram, made from one of Deming's illustrations, will make this clear (Figure I).

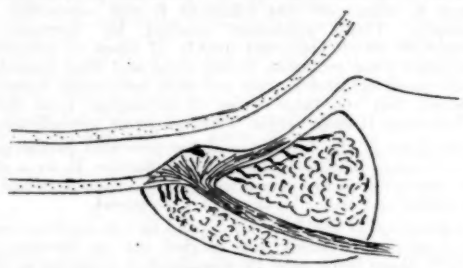

FIGURE I.

Diagram (after Deming) showing the prostatic portion of the urethra, the prostate and the bladder neck (all normal). The prostatic ducts are seen issuing from the masses of prostatic acini to pass through the wall of the urethral floor. Müllerian muscle fibres are seen running along in the wall of the ejaculatory duct to enter the urethral wall and intertwine with the proper plain muscle of that part. The initial lesion of so-called "prostatic" hypertrophy is a fibromyoma arising from the Müllerian muscle.

The second adapted diagrammatic sketch shows the development of such small fibro-myomata in the periurethral region (Figure II). As they grow, they provoke neighbouring epithelium to invade them, and often the invading epithelium overgrows the original fibro-myoma, so that an adenomatous formation results—the second or glandular phase. Deming considers that the epithelium concerned is that of the prostatic ducts, which run through the urethral submucosa to reach the urethra. Such epithelium lines only excretory canals, and is not actually prostatic. Other workers think that the epithelium concerned is that of submucous glands of the urethra. In any case, the net result is that it is most likely that a so-called "prostatic" adenoma is not really prostatic at all, but only periurethral. It is for this reason that Marion, of Paris, has proposed that the word "prostatectomy" be discarded, unless, of course, a radical prostatectomy for carcinoma is concerned, and the name "peri-urethral adenomectomy" be substituted. If one makes a rectal examination after performance of suprapubic "prostatectomy", for instance, one discovers that the prostate gland is still present. While the periurethral neoplasm is growing, the true prostate is simply expanded and compressed, and the plane of surgical cleavage lies between the neoplasm and the true prostate, the latter, thinned out as it is, forming the so-called "surgical capsule" of the neoplasm. It is interesting to note, further, that the second (or glandular) phase of Deming often does not arise at all, for many fairly large "prostates" prove on microscopic examination to be fibro-myomatous all through.

Since the administration of œstrogens or the prevention of androgen formation will retard or even cure a neoplasm arising in an essentially male organ, it might have been expected that androgens would retard, or even cause reduction of, the benign hyperplasias in this region, since they are of female origin. A few years ago much work was done on this aspect, and many reports were published. It is now certain that, from a practical point of view, the medical attendant cannot expect much benefit from the exhibition of androgens in benign hyperplasias, and theoretically this is to be expected. If one knew when early fibro-myomata were present, the administration of androgens might interrupt this tendency; but in practice one has to diagnose obstruction before doing anything at all, and then usually some epithelial elements have entered into play which are not female elements; if they were truly prostatic, which is almost certainly not so, œstrogens would be indicated. The administration of œstrogens, indeed, has been tried, and it was during their trial that several workers discovered the beneficial effects of œstrogens on carcinoma. However, for benign hyperplasias in the prostatic region hormone therapy has been disappointing, and actual removal of the obstruction is necessary.

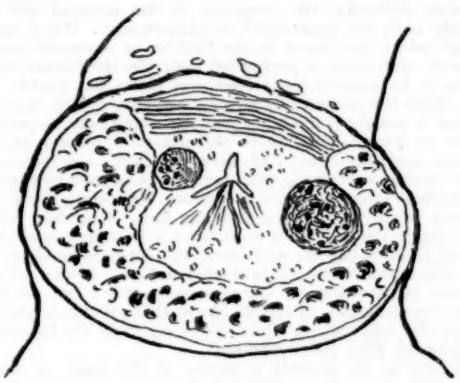

FIGURE II.

Diagram (after Deming) showing a transverse section of the posterior portion of the urethra at the level of the *colliculus seminalis*, with the ejaculatory ducts entering the colliculus. On the left, in the urethral wall, is seen a small fibromyoma, the lateral aspect of which has become invaded by a few glands. On the right is a similar, but larger, new development—an adenofibromyoma. In the further development such new formations become very large, and expand and compress the true prostate against the investing prostatic capsule. Finally, the true prostate becomes a comparatively thin layer, known as the "surgical capsule", because enucleation of an adenoma is performed just within it.

Early Symptomatology.

Early in the development of my experience with the new operation of perurethral prostatic resection, I came to the conclusion that the one point of overwhelming importance is early treatment. It sounds almost too old-fashioned to mention the former habit among practitioners of putting patients off for a few years, by telling them that the growth was not yet "ripe" enough for removal. I do not think that is done much now; but we still see too many patients with prostatic obstruction too late, so that the neoplasm is too large for comfortable or even possible removal through the urethra. It is for this reason that, during the past few years, I contributed two articles here, and one in the United States of America, pleading for early recognition of the symptoms of "prostatism", so that when obstruction was undoubtedly present, endoscopic resection could be used with speed, comfort and safety in every case. That is the ideal, and owing to the fact that patients are actually being sent along for diagnosis quite early in an ever-increasing proportion of cases, this ideal has partly been realized. When the older members of the adult male population learn generally that early prostatic operations are devoid of danger, their own attitude will tend to favour prophylaxis rather than procrastination.[4][5][6]

In the symptomatology of early obstructions, the obstructive aspect has been overshadowed in the past by the greater emphasis placed on irritative symptoms. Severe irritative symptoms, such as excessive frequency of micturition associated with strangury, scalding, bleeding *et cetera*, are expressive of infection and consequent inflammation. These symptoms are not common early in the piece, but unaccustomed frequency of micturition, nocturnal, diurnal or both together, occurs frequently; unfortunately, however, it is sometimes regarded as merely due to old age. This is, of course, incorrect. Signs of obstruction, if noticed at all, are likewise put down to old age, this again being incorrect. The bladder is a hollow muscle, always functioning, and not at all liable to the atony and atrophy of disuse which partially affects the skeletal muscle of older people. A simple proof of this is seen in the amazement with which quite old men regard the strong projection and ample diameter of the urinary stream after they have been relieved of a bladder-neck obstruction. I therefore devised a homely criterion of the presence of restriction of the stream as to force and diameter. This, though rather amusing in its expression, is a practical one, and stringent enough to indicate definite, pathological restriction. It is simply that the patient should be able to project the stream farther than one foot from his toes, and the diameter should be greater than that of an ordinary wooden match. I do not think anyone will quarrel with the stringency of this criterion; actually, of course, some patients with obstructing prostates preserve a moderately wide stream, while the projection is diminished. Many men complain only of the unaccustomed frequency of micturition, which gets them out of bed at night, or prevents them from sitting out a business meeting, but, on questioning, it will be discovered that their projection is only two or even only one and a half feet. Though it is difficult to ascertain such things, since memory is so uncertain, a useful method of questioning is to ask the patient to compare the projection and diameter of his urinary stream with that of ten to twenty years earlier. One is surprised to find how often restriction of the stream antedates increased frequency of micturition.

Occasionally, prostatism is first manifested by a sharp hæmorrhage or sudden retention of urine, seemingly quite "out of the blue", but, on careful questioning, an antecedent history of some degree of restriction of the stream is frequently obtained.

Rectal examination, after tests for residual urine have been carried out, may not disclose any enlargement of the gland, while there may be little or no residual urine. This is of no significance, beyond pointing to an early stage of the development of the neoplasm. If the patient has symptoms, he deserves the benefit of an endoscopic examination with the McCarthy panendoscope. Surprisingly developed obstruction may be seen in the urethra when rectal palpation reveals only a normal surface. The practitioner should seize every occasion to palpate the prostate and check his findings by those of a specialist when the occasion warrants reference to the latter. Prostatic palpation is notoriously difficult from a diagnostic standpoint, and even the most experienced specialist hesitates at times between a diagnosis of carcinoma of the prostate and one of indurated chronic prostatitis. Endoscopic resection, however, as well as providing immediate relief of the obstruction, affords us tissue for biopsy.

Management in General Practice.

If questioning on the foregoing lines fails to elicit a history of stream restriction, but the patient complains of unaccustomed frequency of micturition, one may use an alkali-belladonna mixture. If infection is present, give sulphonamides instead and watch progress. It is wise to have a bacteriological examination made even of macroscopically clear urine. If the condition is resistant to treatment, or if symptoms recur, it is advisable to ask a urologist for advice. In persisting infection, one can carry out a plain X-ray examination with excretion urograms before referring the patient to a specialist. Do not pay too much attention to intraprostatic calculi, grouped behind or just above the *symphysis pubis*, for they are important as a rule only in so far as prostatitis is associated with them. However, a vesical calculus often coexists with bladder-neck obstruction, and more rarely a vesical diverticulum, which may be infected. Remember that a urologist can nearly always crush vesical calculi after removing the prostatic obstruction perurethrally, and so save the patient an open, usually infected, suprapubic wound.

When a patient has much restriction of the urinary stream, more than a few ounces of residual urine, infection, hæmorrhage or frank difficulty in urination, tie in a catheter with tape and strapping. Always use a small catheter, preferably a 7 E *coudé* type; it allows free drainage around itself of urethral seropus, and moreover it is less painful to pass and more comfortable

to retain. This saves time for the patient, and it is the best treatment while communication is being made with a specialist. While the catheter is in place, give the patient one tablet (0·5 gramme) every six hours of some efficient sulphonamide, unless clinical signs of renal insufficiency are present; the dose of sulphonamide can still be given in reduced weight, even down to one-quarter of a tablet every six hours. Experiments at the Mayo Clinic showed that even such small doses have a bacteriostatic effect of practical value.

Acute retention of urine is best dealt with by passing a 7 E or even a 6 E *coudé* catheter, emptying the bladder completely at once, and keeping the patient in bed with the catheter tied in. If not even such small catheters can be passed, one is likely to cause trauma and introduce infection or hæmorrhage into the clinical picture, if one persists with stiffer or larger instruments. I really think it is a more practical procedure then to introduce a fine trocar and cannula, or a serum needle at a pinch, just above the pubis, aiming downwards a little till the instrument enters the bladder, which can then be emptied and the needle withdrawn. After treatment by rest in bed, warmth and the administration of morphine and atropine and a moderate amount of fluids, the patient may pass urine naturally some hours later, or congestion may have decreased enough to allow the passage of the small rubber catheter. I think that every practitioner should keep a 6 E and a 7 E rubber *coudé* catheter in his case. Too often unnecessarily large catheters are used, while these small ones are extremely useful and comfortable for male or female patients. Whenever a catheter is tied in, one should give fluids generously, and continue to give the patient a small maintenance dose of sulphonamide. Irrigations are not really necessary, unless infection or hæmorrhage is present. Then, a simple and cheap routine method is to keep a bottle of 1% silver nitrate solution handy, and use one drachm of this to a pint of hot water (a one in 16,000 solution). This is given as little as possible, but as often as necessary.

In recent years, the attitude to chronic retention of urine has changed somewhat. The patient has a distended bladder; but the distention is painless, and he is often not even aware of the abdominal protuberance. Sometimes dribbling of overflow is present. It is still considered by most people that emptying of the bladder should not be sudden; but a complicated gradual decompression apparatus is not necessary for this purpose. One draws off about 10 to 15 ounces at once, and corks the catheter with a small, conical spigot. If one does not possess this little luxury, simply use a golf tee or the cut-off end of a pen-holder. Do not use an artery forceps or a tape tie; they are clumsy, unpractical and damaging. Every hour during the day, the nurse is instructed to let off eight ounces of urine exactly. This is a few ounces more than the patient secretes, and is an easy way of decompressing the bladder gradually. At the end of thirty-six hours, leave the spigot out altogether, and connect the catheter with glass connexion and tube extension to a Winchester bottle. The catheter should be changed every other day, or every day if this is not too inconvenient. With the relief of chronic retention there is some danger of renal hæmorrhage. It is therefore wise to give the patient as a prophylactic an intravenous injection of 10 cubic centimetres of either a 10% solution of Congo red or a 10% solution of calcium gluconate. If hæmorrhage comes on in spite of this, the injection may be repeated in twelve to twenty-four hours. Sulphonamides are used, as has been indicated, in much reduced dosage, or even not at all, if renal damage is indicated clinically by the presence of a dry tongue or thirst. Drainage is maintained while arrangements are being made to transfer the patient to a specialist.

Management in Special Practice.

As has been mentioned earlier, an ever-increasing proportion of prostatic patients is being referred for specialist treatment at an early stage, and this is a tremendous advantage to all concerned. In such cases, using an instrument of small diameter, the urologist can combine operation with instrumental diagnosis, so that nowadays quite a large proportion of patients enter hospital one evening, have a panendoscopic examination next morning, and can be given the benefit of immediate endoscopic reaction if there is no contraindication. This development has been made possible by means of the small (24 F) resectoscope, this being of the same calibre as the diagnostic panendoscope. In all small and small to moderate obstructions, when no infection or renal deficiency requiring preliminary catheter drainage is present, this is the ideal procedure, and experience shows that there is no danger in it whatever. In fact, in the series of 500 consecutive cases analysed later in this article, not a single death occurred in cases of this smaller type of prostatic obstruction. That is the chief reason why I was so insistent earlier in this article on early diagnosis.

When the patient has more than a few ounces of residual urine, or when the urine is infected, or when there are clinical signs of renal insufficiency or of general ill-health, the whole management is more deliberately planned. A catheter is usually tied in, functional tests of excretion are made each day, the urine is freed of infection, and all necessary studies of systems other than the urinary tract are made. Tests of retention of urine, such as blood urea and blood creatinin tests, are necessary only if the ordinary dye tests of excretion fail to reveal response to drainage.

The establishment of a suprapubic cystostomy for preparation is becoming increasingly uncommon. In the 500 cases analysed later, only 18 preliminary cystostomies were established; four of these patients arrived with the cystostomy already established.

Having cleared the urine as far as is possible, improved the renal state to a reasonable degree, and satisfied oneself about the patient's general condition, one performs a panendoscopic examination. If the instrument will not enter the bladder, or only with difficulty, the condition is the unusual one which definitely calls for suprapubic prostatectomy. When multiple or large calculi are found in the bladder or a vesical neoplasm is present, cystotomy is performed later. In the former case the purpose is to remove the stones and clean the bladder, after which, when the patient has rested for a week or two, endoscopic resection is usually possible, or prostatectomy may be performed at once or later, according to the patient's general and renal condition and the state of bladder infection. Coexistence of a vesical neoplasm is troublesome, and I have learned by experience that the best management is to perform cystotomy, loop off the neoplasm with the electric cutting loop, coagulate bleeding vessels, have the neoplasm implanted with radon seeds by the assisting radium expert, and later, the suprapubic drain being still in place, resect the prostatic obstruction. I have performed this series of procedures in the reverse manner; but the patient is not so comfortable. I should like at this point to acknowledge the splendid cooperation I have received from Dr. Harold Ham in connexion with radon therapy.

It is unwise to proceed with use of the large or standard resectoscope immediately after panendoscopic diagnosis, even though the patient is well prepared, for this instrument is of 28 F calibre, a formidable size. A delay of five days is advisable, after which the urethra dilates kindly up to the required calibre. However, if one possesses a 24 F resectoscope, which I consider a most valuable instrument, even though its resecting loop is somewhat small, one may partially resect the obstruction at the diagnostic sitting, completing the whole operation five days later with the 28 F instrument. I have recently sent a request to Joseph McCarthy, asking him if he would consider getting the "A.C.M.I." firm, of New York, to make a 26 F instrument. The same telescope is used for all these resectoscopes and the diagnostic panendoscope, and the battery of three operating sheaths would be of immense value to any urologist, the cutting loops then being of small, medium and large size.

Should one judge that more than two sessions of resection are necessary to remove all obstructing tissue, it is better to decide on suprapublic prostatectomy, which, performed after the splendid principles of hæmostasis by suture bequeathed to us by the late Harry Harris, is undoubtedly the most perfect form of suprapubic technique in the world today.

My own tendency, however, has been more and more away from prostatectomy during the past ten years. This has been for two main reasons—one, to develop a method which would impose less of an ordeal on the patient, be applicable to a more widely extended range of dangerous lesions and be accompanied by a lower mortality rate, and the other, to gain command of the new technique, which can be accomplished only by performing many hundreds of resections.

Technique of Perurethral Resection and After-Treatment.

The technique of perurethral resection, and the after-treatment, are not of general interest, so I shall be brief over this matter. The object of the operation is to remove all obstructing neoplastic tissue. It is better still to go beyond this, and to remove actually all neoplastic tissue right down to the "surgical capsule", which, as has been explained earlier, is the compressed true prostate. I have drawn a couple of sketches in an attempt to make this clear (Figures III and IV). Legends accompanying these two sketches are self-explanatory; but here I may add a

few points. The great advantage of suprapubic prostatectomy is that the finger, once having entered the proper plane of cleavage, keeps to that plane quite easily, and the innocent neoplasm is totally removed, with practically no risk of recurrence later in life. It does not give protection against the later development of carcinoma, for carcinoma begins as a rule in

FIGURE III.

A trilobed "prostatic" adenoma shown semi-diagrammatically in a composite panendoscopic view.

the true prostate, which, as has been explained earlier, is not removed at all at the ordinary "prostatectomy". On the other hand, the smaller, fibrous types of obstruction, together with purely meatal types of obstruction, have not easy planes of cleavage, and are more suited to endoscopic resection as the "operation of choice" in the stricter or obligatory sense. The

FIGURE IV.

Same case as Figure III, towards the completion of an endoscopic resection. The middle lobe has been completely resected, the cut distal edge of the trigonal muscle being exposed; this is seen with a dark shadow under it to indicate a shallow sulcus. The right lateral lobe has been almost completely resected. The lumpy tissue is adenoma not completely resected from the underlying fairly smooth but coarsely striated tissue, which is the compressed true prostate or "surgical capsule".

difficulty in perurethral resection is to be able to recognize visually the various tissues encountered, so that on the one hand all neoplastic tissue is removed, while on the other hand the planes of safety are not transgressed. Only an inexperienced operator will perforate the trigone itself or bladder wall behind the internal meatal region; but the danger is ever-present for even the most experienced in the meatal region itself. When the neoplastic tissue has been carved in strips from the trigonal muscle, the edge of the latter is seen as quite a thick ledge at the internal meatus. Under the ledge is a shadow. It is better not to undermine the trigone at all, though this is done in suprapubic prostatectomy. The enucleating finger, however, is not so dangerous as the electric cutting loop. Throughout the mass of the neoplastic tissue it is a matter of cutting away lumpy, granular adenoma or other types of tissue until one comes to the compressed true prostate, which has a coarsely and irregularly striated appearance, contrasting strongly with the lumpy neoplastic tissue. The trigonal edge, too, is striated, but more finely. The neoplastic tissue has to be removed all around the floor, lateral walls and roof of the urethra, from the proximal edge of the intravesical protrusion right out to include the distal extremities of the lateral "lobes" of the neoplasm. The latter extend to, and usually a little beyond, the *colliculus seminalis*. Just distal to the colliculus the voluntary sphincter of the membranous urethra lies encircling the canal, and it must be held sacrosanct and kept inviolate, otherwise incontinence of urine will result. The margin of safety is small, and the most experienced operators have experienced trouble at this point. The method I have come to adopt, to render this perfectly safe, is to aim the instrument like a pistol obliquely at the colliculus, so that water pressure drives the distal tips of the lateral lobes apart, disclosing the position of the colliculus clearly between each cutting of a strip from the lateral lobes.

The other important point in technique is hæmostasis. This is effected by switching over to the coagulating current and touching the spurting vessel at the exact point where the little fountain of blood issues from the tissue. I consider it essential to secure absolute or as nearly as possible absolute hæmostasis. Many warnings have been issued in the United States of America by leading urologists against what they call over-coagulation. I agree that one should not coagulate tissue indiscriminately; but I do think that all bleeding vessels should be coagulated. The risks of too much coagulation are supposed to be continuing infection and sometimes secondary hæmorrhage. Now, surely, the risk of secondary hæmorrhage is greatest from separation of a slough from a point overlying an artery or arteriole; but such a point will bleed strongly during the resection, and simply must be coagulated. All other areas are less dangerous as regards risk of secondary hæmorrhage. If partially detached flaps of tissue are left behind, urination drives them open, and may cause hæmorrhage. Such flaps also cause continuing sepsis. The great thing in resection is to remove all obstructing tissue so that the patient has a powerful, unobstructed stream. Any turbidity of the urine that remains for some weeks, or even a month or so, will be of no disadvantage to the patient provided he has no frequency or scalding of micturition, and passes a free stream. There is more risk of secondary hæmorrhage from incomplete removal of tissue than from adequate coagulation over an arteriole. I disagree entirely with many urologists in the United States of America, who send their patients back to the ward with blood in the irrigating fluid. Some clinics evidently consider loss of blood during and after resection as a necessary evil, and give blood transfusion as a routine measure. One clinic, one of the birth-places of endoscopic resection, orders syringing and clot evacuation every fifteen minutes for several hours as the initial phase of after-treatment. Surely endoscopic resection is not justified unless, among manifold other advantages, it spares the patient loss of blood and renders the nursing infinitely easier than after prostatectomy. I consider that hæmostasis should be progressive and absolute throughout the whole operation, and that, except in unlucky instances, hæmorrhage should be only trifling afterwards. My experience has been that the more assiduously I have coagulated bleeding vessels, the less reactionary and secondary hæmorrhage I have had.

The after-treatment is simple; it is just a matter of preventing the retained catheter from becoming blocked. There is practically no shock associated with the operation, and no after pain unless the catheter becomes blocked. I tie in a 9, 10 or 11 E *coudé* catheter for four to six days, according to the magnitude of the resection performed. Usually the patient can get out of bed in two or three days, walk about on the fourth or fifth day, and leave hospital about three days after the catheter has been removed—that is, seven to nine days after the operation. We can add four or five days to this if the resection has to be done in two sessions.

Biopsy and examination of tissue ideally are performed in every case; but they are obligatory when the slightest suspicion of malignancy is obtained on palpation. In this case, the pathologist is asked to push the examination through urgently in a few days. If the tissue is malignant, it is explained to the patient, euphemistically, that a dangerous development will occur in the prostate unless he undergoes a small operation consisting in the removal of "portion" of each testicle. This is subcapsular orchidectomy, in which the seminiferous tubules with interstitial tissue are alone removed; the epididymis and the two tunicæ are left behind, and they feel like a small testicle afterwards and so obviate mental worry over the matter. If this is not done, the patient has to undergo the inconvenience of seeing his doctor twice a week for ever, for intramuscular injections of stilbœstroldipropionate, and take the same substance by mouth as well. Removal of the testicular tissue has a continuous, smooth and permanent effect, only a small tablet of stilbœstroldipropionate (one to two milligrammes) by mouth once a day being necessary to counteract androgen formation by the suprarenal glands.

Analysis of Cases.

Of 500 consecutive patients with bladder-neck obstruction treated during a little under five years up to the end of 1942, 468 were treated for their obstruction by perurethral resection, except one woman who had over-dilatation instead. There were actually four cases among females, two of whom had complete retention of urine. All patients were relieved of their obstruction in this series of 468 resections, except one male, who went elsewhere and was found, when the bladder was opened, to have a carcinoma of the prostate, and another, with an early lesion, on whom I had to perform suprapubic prostatectomy when resection failed to relieve him. Three permanent cystostomies were established for prostatic carcinoma, and one patient, aged forty-three years, had suprapubic and perineal implantation of radon seeds in a futile attempt to influence the course of a spindle-cell sarcoma of the prostate. Twenty-four patients only in the whole series were treated by suprapubic prostatectomy.

Of this series of 468 resections, in 48 cases microscopic examination proved the condition to be carcinomatous. Eighteen patients had a preliminary cystostomy established before their resection. One case was complicated by malignant neoplasm of the bladder, on account of which open operation was performed later, one was complicated by a heavily infected vesical diverticulum, the open operation of diverticulectomy being performed later, and one was followed by the establishment of a suprapubic cystostomy to implant radon seeds, when the prostate condition, thought to be innocent, was found to be malignant.

Morbidity.

The morbidity due to the operation of endoscopic resection included three cases of incontinence of urine. One patient was completely relieved by a perineal plastic repair operation, another was only partially relieved by the same measure, while the third is so comfortable with a penile incontinence clamp that he refuses plastic repair. In this series, up to the end of 1942, I experienced two cases of rupture of the bladder at the internal meatal edge. One patient died because neither he nor his family would consent to any form of open operation for either the disease (advanced carcinoma) or the complication. The other patient underwent suprapubic and extravesical drainage, and recovered. There were four cases of stricture of the urethra, in which a rigid urethra was traumatized by passage and manipulation of the resectoscope. If a resectoscope will not move freely in and out of the urethra, it is better to perform suprapubic operation. All of these stricture patients have been relieved by dilatation.

Mortality.

Some years ago, an American urologist analysed a series of all cases of bladder-neck obstruction encountered during a certain period of years, and found that the mortality rate of prostatism was shockingly high. Some patients died before any treatment at all could be given, others after the simple passage of a catheter, while still others died after cystoscopic examination. This surgeon found that the mortality rate of suprapubic cystostomy was relatively enormous. When all these were subtracted, one commenced to calculate the mortality rate of prostatectomy and of endoscopic resection.

In the period of my series, I have not been able to go into the matter of deaths occurring before any kind of operation. Neither have I studied the remote mortality rate; this, however, needs careful analysis to be of any value, for most deaths within a few years of prostatic operations would be found to be due to unconnected causes. It is certain, however, that one of the great advantages of perurethral resection is its wider applicability to the most dangerous group of cases. My impression during the period under study is that very few risks were unaccepted.

In the series of 24 suprapubic prostatectomies, one death occurred (a mortality rate of about 4%). In the series of 468 perurethral resections, seven deaths occurred (a mortality rate of about 1·5%). In the remaining odd types of case, no deaths occurred.

Finally, it is interesting to note that in the three cases of permanent cystostomy for carcinoma, and in the 18 cases of preliminary cystostomy, no death occurred. As has been stated earlier, this particular operation used to have a formidable mortality rate because of the reaction in septic or uræmic patients. I now consider it better in such cases to avoid cystostomy and to perform a small "try-out" resection, proceeding to completion later. It would be interesting to give details of such dangerous cases and their management, but space restrictions forbid this.

Conclusions.

In recent years an amazing revolution has been witnessed in the treatment of bladder-neck obstructions, innocent as well as malignant—namely, the perurethral attack on all obstructions, with antiandrogen therapy for malignant ones.

The medical profession and the public should become more conscious of the early symptomatology of bladder-neck obstruction, with the object of securing early and therefore full benefit from these new therapeutic procedures.

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THE PRECIPITIN, AGGLUTINATION, INDOLE AND METHYL RED REACTIONS OF THE DYSENTERY BACILLI.

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THE precipitin test has proved valuable for dividing the hæmolytic streptococci into groups, of which four (A, B, C and G) contain the majority of types that are pathogenic for man. This test is simple and rapid to perform and is economical in requirements.

The use of the precipitin test for the identification of the dysentery bacilli has not received much attention, but the biochemical and agglutination reactions have been thoroughly studied. The "sugar" reactions provide a ready means of dividing these organisms into three classes, namely: (*a*) those which do not ferment mannitol (Shiga and Schmitz types), (*b*) those which ferment mannitol and which ferment lactose late (Sonne type), and (*c*) those which ferment mannitol but do not ferment lactose (Flexner and Boyd types).

The first two classes have other differences, such as indole production, which simplify the classification of the individual types. The third class contains many types, most of which have the same biochemical reactions and morphological characteristics. Two different classifications of this class have been presented. The first is generally referred to as the Andrewes and Inman classification and the second as that of Boyd.

The Andrewes and Inman classification divides the class which ferments mannitol but does not ferment lactose into types, each of which possesses the four antigens V, W, X and Z. An organism is named by the antigen which preponderates. A fifth type, the Hiss-Russell Y, contains the V, W and Z antigens in nearly equal amounts, together with a small amount of the X component (Topley and Wilson).[1] The classification is based on an extensive series of cross-agglutination and reciprocal absorption tests (Gardner).[2] There is no suggestion that the different types possess specific antigens of their own.

Boyd[3] divides this class into two groups—the Flexner and Boyd groups. The Flexner group is subdivided into six types, each possessing a type-specific antigen and an antigen common to all types of this group—that is, a group antigen. Minor antigens are also present, and some of these are shared. The cross-agglutination among the members of this group is due to the reaction between the common group antigen and the corresponding antibody. The organisms are typed by agglutination tests with monospecific antisera, which are antisera prepared in the usual manner with the different type organisms, but from which the common group antibody has been absorbed. Boyd found that the Hiss-Russell Y strain possessed abundant group antigen, but was devoid of any type-specific antigen, and was therefore ideal for absorbing the group antibody from the type antisera. However, the presence of the shared minor antigens and the corresponding antibodies in the antisera made it necessary to absorb these antibodies if an absolutely monospecific antiserum was to be prepared. These were absorbed by Flexner type X and Boyd types 103B and P119B, the last two organisms being variants which possess no type-specific antigen. Using a complete set of monospecific antisera, one may type an organism by slide agglutination.

The Boyd group of the class which ferments mannitol but not lactose comprises types which possess type-specific antigens but have no common group antigen. The main members of this group are the four Boyd types—I, II, III and P274. Other organisms have been placed in this group; but their pathogenicity for man is not conclusive.

The degree of cross-agglutination which takes place when type antisera which are not monospecific are used, renders the agglutination test of limited value for typing the Flexner organisms. A test detecting the major "type" antigens only would be of great service. Such a test is that using the monospecific antisera; but if all the necessary absorbing types are not available, the satisfactory preparation of these monospecific antisera is not possible. With the simple precipitin method for identifying the groups of the hæmolytic streptococci in mind, the possibility of such a test for typing the Flexner group of dysentery bacilli was investigated. Many workers, including P. B. White,[4] Ando[5] and Morgan[6] have demonstrated the presence of soluble specific substances on the intestinal bacilli, and the method of extraction used in this work resembled that of Morgan.

EXPERIMENTAL WORK.

The principal object of this work was to devise a precipitin test for typing the Flexner group of organisms and to compare its value with that of the agglutination reaction. The investigation also included tests with other dysentery types and a heterogeneous group of organisms related to them. The latter were *Bacterium alkalescens*, which is serologically related to Boyd type P274, and *Bacterium dispar*, which is a late fermenter of lactose very similar to the Sonne type. Flexner types X and Y, whose classification is doubtful, were also included.

The organisms used were the following: Flexner types I to VI, Boyd types I, II, III and P274, Flexner types X and Y (Hiss-Russell strain), Sonne, Schmitz and Shiga strains; *Bacterium alkalescens* and *Bacterium dispar*. They were all standard type cultures.

The antisera were those corresponding to the foregoing organisms, with the exception of Flexner type X, *Bacterium alkalescens* and *Bacterium dispar*. Antisera of the Salmonella group (*Bacterium typhosum* and *Bacterium paratyphosum A*, *B* and *C*) were also included.

Organisms and antisera were supplied by the Commonwealth Serum Laboratories. The nomenclature used for the Flexner types I to VI and the Boyd types is that given in the Boyd[3] classification.

Indole and methyl red tests were included to determine their value for identification of the types.

Methods.

Preparation of Thick Suspensions.

Thick suspensions were prepared as follows.

Cultures of smooth organisms were grown on nutrient agar in Petri dishes for eighteen hours at 37° C. The growth was washed off with saline solution and the suspension was centrifuged until the supernatant fluid was clear. This required from one-half to one hour at 2,000 to 3,000 revolutions per minute. The supernatant fluid was discarded and the deposit was suspended in 5·0 millilitres of normal saline solution. The strength of the suspension was determined by the opacity method. A thickly inoculated Petri dish provided 50,000 to 60,000 million organisms and an agar slope in a test tube measuring six inches by three-quarters of an inch provided from 20,000 to 30,000 million organisms. For the tests described suspensions containing 100,000 to 150,000 million organisms were prepared.

For the agglutination tests, 40,000 million organisms were pipetted into a centrifuge tube and two volumes of alcohol were added. This suspension was incubated overnight at 37° C. and on the following day was centrifuged; the supernatant fluid was discarded, and one millilitre of 33% alcohol was added. In this state the suspension was stored in the refrigerator. When required the one millilitre of alcoholic suspension was poured into 39 millilitres of sterile normal saline solution, the final strength of the suspension thus being brought to 1,000 million organisms per millilitre.

The remainder of the thick suspension was used for preparing the extracts for the precipitin tests.

Technique of Performing the Agglutination Tests.

The agglutination tests were performed as follows.

An amount of 0·2 millilitre of the suspension already described for the agglutination tests was added to 0·2 millilitre of the serial dilutions of the antisera in test tubes three inches by three-eights of an inch in size. The tubes were placed in the 52° C. water bath for two hours, after which period they were removed and left at room temperature overnight. Results were read, a hand lens being used to detect fine agglutination.

The quantities of reagents were the smallest which provided an easily readable test. Test-tubes were used in preference to standard type agglutination tubes because they were easier to clean, and efficient mixing was obtained by a few vigorous shakes. Positive reactions occurring in dilutions lower than one in 320 are not recorded in this report.

Preparation of the Extract for the Precipitin Test.

The extract for the precipitin test was prepared in the following manner.

The remainder of the thick suspension was diluted with saline solution to a strength of 10,000 million organisms per millilitre. Methyl red was added as an indicator, and the suspension acidified with N/5 acetic acid solution until a definite red colour was obtained. Some organisms—particularly Flexner types IV and VI—reduce the dye and render it useless as an indicator. In this case the dye can be used as an outside indicator. The test tube containing the suspension was stoppered and placed in a boiling water-bath for half an hour, after which period it was removed and allowed to cool. When it was cool, phenol red indicator was added and the reaction was brought to neutrality with N/5 sodium hydroxide solution. The suspension was centrifuged for thirty to sixty minutes to obtain an absolutely clear supernatant fluid. This supernatant fluid—henceforth called the extract—was decanted into a suitable container and stored in the refrigerator until required.[1]

Dilutions of Antisera for the Precipitin Test.

The antisera were diluted to one in 10 with the exception of Flexner types I and II. These were diluted to one in 40 to give better differentiation.

Technique of Performing the Precipitin Tests.

Amounts of 0·2 millilitre of extract were added to 0·2 millilitre of the diluted antisera in test-tubes measuring three inches by three-eighths of an inch, which were placed in the 52° C. water-bath for one hour and then examined. In the recording of the results, four degrees of reaction were recognized: (i) precipitation which was visible with the naked eye; (ii) precipitation

[1] For the identification of recently isolated strains sufficient extract can be prepared by washing off the growth from two agar slope cultures with 5·0 millilitres of normal saline solution. This suspension is then acidified, as above, without previous washing.

which was visible with a hand-lens (approximately ×4); (iii) thick opalescence; (iv) opalescence, which although not so conspicuous as the previous class, was easily detected when compared with the saline control. Adequate controls were included.

The Test for Indole.

To detect indole, Ehrlich's reagent was layered on twenty-four and forty-eight hour cultures of the organisms in a 1% solution of peptone in 0·5% saline solution. With good indole producers a red ring appeared at the junction of the two liquids, whereas if no definite ring appeared the result was regarded as negative. This avoided the recording of the doubtful reactions given by indifferent indole producers after prolonged culturing. Some of the organisms do not grow rapidly in this medium, and for this reason a test was made on the second day.

The Methyl Red Test.

The methyl red test was performed in the usual way on cultures incubated for one and two days.

Results Obtained with Standard Type Cultures.[1]

Flexner Type I.

With Flexner type I antiserum the extract formed a precipitate which was visible with the hand lens; with Flexner type II antiserum a mild opalescence was obtained. These were the only reactions to the precipitin test. Flexner type I extract was agglutinated by a wide variety of antisera. It was agglutinated to significant titre by the antisera of Flexner types I to VI and Y, by Boyd types II and P274, by *Bacterium paratyphosum A* and *C* and to a lesser degree by Sonne and *Bacterium typhosum* antisera. Flexner type I cultures failed to react to the test for indole and to the methyl red test.

Flexner Type II.

When the precipitin test was applied, Flexner type II extract reacted with Flexner type II antiserum only, and gave an easily visible precipitate. The organism was not agglutinated by Flexner type III antiserum, but tests with the antisera of the other Flexner types, with Boyd types II and P274, with *Bacterium paratyphosum A* and *C* and with *Bacterium typhosum* gave positive results. Flexner type II cultures failed to react to the test for indole and to the methyl red test.

[1] The precipitin reactions have been summarized in Table I and the agglutination reactions in Table II.

Flexner Type III.

When the precipitin test was applied, Flexner type III was the least reactive of all the Flexner types. The extract gave an opalescence with its own antiserum, but did not react with any of the others. With regard to the agglutination test, Flexner type VI antiserum did not agglutinate Flexner type III, which was agglutinated by the other Flexner type antisera as well as by that of Boyd type P274. No agglutination was obtained with the Salmonella antisera.

The cultures yielded a reaction on the first day to the test for indole, but failed to react to the methyl red test.

Flexner Type IV.

When the precipitin test was applied, Flexner type IV extract gave a heavy opalescence with the antisera of Flexner types II and IV and visible opalescence with those of Flexner types I, III, V, VI, Y and Boyd type P274. Flexner type IV was agglutinated by the antisera of all Flexner types, and by Boyd types II and P274. It was not agglutinated by the Salmonella antisera. The culture on the first day yielded a reaction to the test for indole. The culture failed to react to the methyl red test on the first day, but reacted on the second day.

Flexner Type V.

When the precipitin test was applied, Flexner type V extract gave an obvious precipitate with its own antiserum and a mild opalescence with that of Boyd type P274. The antisera of all the Flexner types except type VI, of Boyd type P274 and of *Bacterium paratyphosum A* and *C* agglutinated Flexner type V. The organism usually, but not always, gave a positive reaction to the test for indole on the first day; the reaction was undoubtedly positive on the second day. The organism failed to react to the methyl red test.

Flexner Type VI.

Flexner type VI extract gave an obvious precipitate with its own antiserum, but did not react with any other. Flexner type VI was agglutinated by Flexner types II and VI antisera only. It was not agglutinated by the Salmonella antisera. The culture failed to react to the test for indole. The organism reacted to the methyl red test on the first day.

TABLE I.
Results of the Precipitin Test.

Organism Extract.	Antisera (with Dilutions Used).[1]													
	Flexner Type I (1 in 40)	Flexner Type II (1 in 40)	Flexner Type III (1 in 10)	Flexner Type IV (1 in 10)	Flexner Type V (1 in 10)	Flexner Type VI (1 in 10)	Flexner Type Y (1 in 10)	Boyd Type I (1 in 10)	Boyd Type II (1 in 10)	Boyd Type III (1 in 10)	Boyd P274 (1 in 10)	Sonne Type (1 in 10)	Schmitz Type (1 in 10)	Shiga Type (1 in 10)
Flexner type I ..	P[2]	o[2]	—	—	—	—	—	—	—	—	—	—	—	—
Flexner type II ..	—	PP[2]	—	—	—	—	—	—	—	—	—	—	—	—
Flexner type III ..	—	—	o	—	—	—	—	—	—	—	—	—	—	—
Flexner type IV ..	o	O[2]	o	O	o	o	o	—	—	—	o	—	—	—
Flexner type V ..	—	—	—	—	PP	—	—	—	—	—	o	—	—	—
Flexner type VI ..	—	—	—	—	—	PP	—	—	—	—	—	—	—	—
Flexner type X ..	—	P	—	—	—	—	—	—	—	—	—	—	—	—
Flexner type Y ..	o	P	P	PP	P	P	PP	—	—	—	—	—	—	—
Boyd type I ..	—	—	—	—	—	—	—	PP	—	—	—	—	—	—
Boyd type II ..	—	—	—	—	—	—	—	—	PP	—	—	—	—	—
Boyd type III ..	—	—	—	—	—	—	—	—	—	P	—	—	—	—
Boyd type P274 ..	—	—	—	—	—	—	—	—	—	—	PP	—	—	—
Sonne type	—	—	—	—	—	—	—	—	—	—	—	O	—	—
Schmitz type ..	—	—	—	—	—	—	—	—	—	—	—	—	O	—
Shiga type	—	—	—	—	—	—	—	—	—	—	—	—	—	O
Bacterium alkalescens	—	—	—	—	—	—	—	P	—	—	o	—	—	—
Bacterium dispar ..	—	—	—	—	—	—	—	—	—	—	—	—	—	—

[1] Results were read after one hour in the 52° C. water-bath.
[2] Abbreviations used: "PP" = precipitate easily visible; "P" = precipitate visible with the aid of a hand-lens; "O" = heavy opalescence; "o" = visible opalescence.

TABLE II.
Results of Agglutination Tests.[1]

Organism.	Antisera.																	
	Flexner Type I.	Flexner Type II.	Flexner Type III.	Flexner Type IV.	Flexner Type V.	Flexner Type VI.	Flexner Type X.	Boyd Type I.	Boyd Type II.	Boyd Type III.	Boyd Type P274.	Sonne Type.	Schmitz Type.	Shiga Type.	*Bacterium paratyphosum A* (1,280).[2]	*Bacterium paratyphosum B* (1,280).[2]	*Bacterium paratyphosum C* (2,560).[2]	*Bacterium typhosum* (5,120).[2]
Flexner type I ..	20,480	10,240	320	1,280	1,280	1,280	2,560	—	1,280	—	2,560	320	—	—	640	—	1,280	320
Flexner type II ..	2,560	40,960	—	320	320	640	640	—	640	—	1,280	—	—	—	320	—	640	1,280
Flexner type III ..	2,560	2,560	5,120	320	1,280	—	640	—	—	—	640	—	—	—	—	—	—	—
Flexner type IV ..	5,120	10,240	1,280	10,240	640	1,280	5,120	—	640	—	640	—	—	—	—	—	—	—
Flexner type V ..	2,560	640	640	1,280	5,120	—	1,280	—	—	—	5,120	—	—	—	320	—	640	—
Flexner type VI ..	—	320	—	—	—	1,280	—	—	—	—	—	—	—	—	—	—	—	—
Flexner type X ..	5,120	40,960	640	1,280	2,560	—	640	—	—	—	2,560	—	—	—	640	—	1,280	640
Flexner type Y ..	5,120	10,240	2,560	10,240	2,560	2,560	5,120	—	640	—	1,280	—	—	—	—	—	—	—
Boyd type I	—	—	—	—	—	—	—	1,280	—	—	—	—	—	—	—	—	—	—
Boyd type II	—	—	—	—	—	—	—	—	2,560	—	—	—	—	—	—	—	—	—
Boyd type III	—	320	—	—	—	—	—	—	—	2,560	—	—	—	—	—	—	—	—
Boyd type P274 ..	320	640	—	—	—	—	—	320	—	—	2,560	—	—	—	320	—	—	1,280
Sonne type	320	—	—	—	—	—	—	—	640	—	—	1,280	—	—	—	640	—	—
Schmitz type	—	—	—	—	—	—	—	2,560	—	—	—	—	5,120	—	—	—	—	—
Shiga type	—	640	—	—	—	—	—	—	—	—	320	—	—	5,120	—	—	—	—
Bacterium alkalescens ..	1,280	5,120	—	320	—	—	320	5,120	640	—	1,280	—	—	640	—	—	—	—
Bacterium dispar ..	640	—	—	1,280	320	320	—	—	—	—	320	—	—	—	—	—	320	—

[1] Results were read after two hours in the 52° C. water-bath and overnight at room temperature. Only titres of one in 320 or over are recorded.
[2] These figures represent the "O" titres of the antisera.

Flexner Type X.

When the precipitin test was applied, Flexner type X extract reacted with Flexner type II antiserum only. Flexner type X was agglutinated by the antisera of all the Flexner types except type VI, and by those of Boyd type P274, *Bacterium paratyphosum A* and *C* and *Bacterium typhosum*. The organism failed to react to the test for indole. The organism did not react on the first day to the methyl red test, but did react on the second day.

Flexner Type Y (Hiss-Russell Y).

Flexner type Y extract formed precipitates with all the Flexner types except type I. With this antiserum an opalescence was formed. Flexner type Y was agglutinated by the antisera of all the Flexner types, and by those of Boyd types II and P274. The cultures failed to react to the test for indole and to the methyl red test.

Boyd Type I.

Boyd type I extract was precipitated by its own antiserum, but did not react with any of the others. The organism was agglutinated by its own antiserum only. The culture failed to react to the test for indole. The methyl red test produced a positive reaction on the first day.

Boyd Type II.

Boyd type II extract reacted only with the homologous antiserum, with which it formed an easily visible precipitate. The agglutination test produced a positive result with the homologous antiserum only. Boyd type II culture failed to react to the test for indole; but it reacted on the first day to the methyl red test.

Boyd Type III.

Boyd type III extract gave a precipitate with the homologous antiserum, but did not react with any of the other antisera. When the agglutination test was applied, apart from a weak agglutination with Flexner type II antiserum, the homologous antiserum was the only one to give a reaction. The organism failed to react to the test for indole, but on the first day yielded a positive reaction to the methyl red test.

Boyd Type P274.

When the precipitin test was applied to Boyd type 274 extract, a heavy precipitate was formed with the homologous antiserum; this was the only reaction. The organism was agglutinated to a significant titre by its own antiserum and by those of *Bacterium paratyphosum A* and *Bacterium typhosum*. It also gave weak agglutination with Flexner types I and II and Boyd type I antisera. The organism failed to react to the test for indole, but reacted on the first day to the methyl red test.

Sonne Type.

With the Sonne type of organism the precipitin test produced heavy opalescence with the homologous antiserum, but the others gave no reaction. The organism was agglutinated by the homologous antiserum and by those of Flexner type I, Boyd type II and *Bacterium paratyphosum B*. The organism failed to react to the test for indole, but gave a reaction on the first day to the methyl red test.

Schmitz Type.

When the precipitin test was applied to the Schmitz type of organism, the extract gave a heavy opalescence with its own antiserum, but otherwise no reaction was noted. The organism was agglutinated by its own antiserum and by that of Boyd type I. The cultures yielded positive reactions on the first day to the test for indole and to the methyl red test.

Shiga Type.

When the precipitin test was applied to the Shiga type of organism, the extract gave a heavy opalescence with its own antiserum, but did not react with any of the others. The organism was agglutinated by its own antiserum, but gave only weak agglutinations with Flexner type II and Boyd type P274 antisera. The organism failed to react to the test for indole and to the methyl red test.

Bacterium alkalescens.

When the precipitin test was applied to *Bacterium alkalescens*, the extract gave a heavy opalescence with Boyd type II and a mild opalescence with Boyd type P274. With the latter antiserum the difference in reaction between the extract of this organism and the extract of Boyd type P274 was pronounced. *Bacterium alkalescens* was agglutinated by many of the antisera—to a significant degree by Flexner type II, Boyd types I and P274 antisera and to a lesser extent by Flexner types I, IV and Y, Boyd type II and the Shiga type antisera. The organism reacted on the first day to the test for indole and to the methyl red test.

Bacterium dispar.

When the precipitin test was applied to *Bacterium dispar*, the extract gave no reaction with any of the antisera. The organism was agglutinated to a mild degree by the antisera of Flexner types I, IV, V, VI, Boyd type P274 and *Bacterium paratyphosum C*. It reacted on the first day to the test for indole and to the methyl red test.

Results of Experimental Work with Locally Isolated Strains.

The tests were applied to a collection of dysentery bacilli isolated from cases at this hospital during the twelve months ending June 30, 1943. The Flexner organisms were typed by slide agglutination with Flexner type antisera which had been absorbed with a Flexner type Y culture obtained from the bacteriological department of the University of Adelaide. The reactions of this organism (called YS) are given at the end of this section. The collection consisted of 23 strains of Flexner type II, two strains of Flexner type IV, two strains of Flexner type V and 14 strains of the Sonne type. The reaction of the standard type organism is given to make possible comparison with the reactions of the local strains.

The Precipitin Test.

Flexner Type II.—The reaction to the precipitin test of the standard type of Flexner type II is a precipitate with Flexner type II antiserum only. Twenty-three local strains were tested; 14 gave a precipitate with Flexner type II antiserum only, and nine gave a precipitate with Flexner type II antiserum and a trace of opalescence with Flexner type I antiserum.

Flexner Type IV.—The reaction to the precipitin test of the standard type of Flexner type IV is opalescence with Flexner types I to VI and Boyd type P274. Two local strains were tested; both were isolated from the same patient, the second strain three days after the first. The first strain, A5, gave opalescence with Flexner types I to VI and Y antisera. The second strain, A6, gave a precipitate with Flexner types II, IV and Y antisera. Opalescence was obtained with Flexner types I, III and V and with Boyd types I and P274.

Flexner Type V.—The reaction to the precipitin test of the standard type of Flexner type V is a precipitate with Flexner type V antiserum only. Two local strains were tested; both gave the same reaction as the standard type of the organism.

Sonne Type.—The reaction to the precipitin test of the standard Sonne type is heavy opalescence with Sonne type antiserum. Fourteen local strains were tested; 13 gave heavy opalescence with Sonne type antiserum and one gave visible opalescence with Sonne type antiserum.

The Agglutination Tests.

Because only two strains of Flexner types IV and V were available, the examination of the other types was reduced to this number. The strains chosen were the first and last to be isolated in each case. The reactions are arranged in table form and the reaction of the standard type is given for comparison.

Test for Indole and the Methyl Red Test.

For the indole and methyl red tests all strains available were used. Standard type reactions are also given.

Flexner YS.

Flexner YS was a strain of Flexner Y obtained from the bacteriological department of the University of Adelaide. It originally came from Boyd, who sent it to Dr. Burnet of Melbourne, who in turn sent it to Adelaide. The reactions of this organism are given because they are mentioned in the discussion on the classification of the Flexner type Y organism.

TABLE III.
Flexner Type II.

Antiserum.	Standard Type.	Local Strains.	
		A1.	A46.
Flexner type I ..	10,240	10,240	5,120
Flexner type II ..	40,960	40,960	40,960
Flexner type III ..	—	—	320
Flexner type IV ..	320	1,280	1,280
Flexner type V ..	320	640	320
Flexner type VI ..	640	1,280	1,280
Flexner type Y ..	640	1,280	1,280
Boyd type II	640	640	640
Boyd type P274 ..	1,280	1,280	1,280
Bacterium paratyphosum A	320	1,280	640
Bacterium paratyphosum C	640	1,280	1,280
Bacterium typhosum ..	1,280	2,560	2,560

TABLE IV.
Flexner Type IV.

Antiserum.	Standard Type.	Local Strains.	
		A5.	A6.
Flexner type I ..	5,120	10,240	10,240
Flexner type II ..	10,240	10,240	10,240
Flexner type III ..	1,280	2,560	2,560
Flexner type IV	10,240	10,240	10,240
Flexner type V ..	640	1,280	1,280
Flexner type VI ..	1,280	1,280	1,280
Flexner type Y ..	5,120	5,120	5,120
Boyd type II	640	1,280	1,280
Boyd type P274	640	2,560	2,560

TABLE V.
Flexner Type V.

Antiserum.	Standard Type.	Local Strains.	
		A42.	A43.
Flexner type I ..	2,560	2,560	1,280
Flexner type II ..	640	1,280	1,280
Flexner type III ..	640	1,280	640
Flexner type IV ..	1,280	2,560	640
Flexner type V ..	5,120	10,240	5,120
Flexner type VI ..	—	320	—
Flexner type Y ..	1,280	1,280	320
Boyd type P274	5,120	5,120	2,560
Bacterium paratyphosum A	320	640	—
Bacterium paratyphosum C	640	1,280	640
Bacterium typhosum ..	—	640	—

TABLE VI.
Sonne Type.

Antiserum.	Standard Type.	Local Strains.	
		A20.	A45.
Flexner type I ..	320	640	640
Flexner type II ..	—	—	320
Flexner type III ..	—	—	320
Boyd type II	640	—	320
Sonne type	1,280	2,560	2,560
Bacterium paratyphosum B	640	320	1,280
Bacterium typhosum ..	—	320	320

The organism gave a positive reaction to the test for indole. The methyl red test produced no reaction on the first day and a positive reaction on the second.

DISCUSSION.

The major antigens of the Flexner types of dysentery bacilli are present either as specific type antigens (Boyd hypothesis) or as the predominant antigen of a series of shared antigens (Andrewes and Inman hypothesis). By a simple extraction with acid the active principle of these major antigens can be

TABLE VII.

Organism.	Indole.	Methyl Red.	
		One Day.	Two Days.
Flexner type II (standard)	−	−	−
Flexner type II (7 strains)	−	−	−
Flexner type II (16 strains)	−	−	+
Flexner type IV (standard)	+	−	+
Flexner type IV (1 strain)	−	−	+
Flexner type IV (1 strain)	−	+	+
Flexner type V (standard)	+	−	−
Flexner type V (1 strain)	+	−	−
Flexner type V (1 strain)	+	+	+
Sonne type (standard)	−	+	+
Sonne type (14 strains)	−	+	+

TABLE VIII.

Antiserum.	Agglutination Titre.	Response to Precipitin Test.
Flexner type I	5,120	Opalescence (mild).
Flexner type II	2,560	Opalescence (heavy).
Flexner type III	—	Opalescence (mild).
Flexner type IV	1,280	Opalescence (heavy).
Flexner type V	—	Opalescence (mild).
Flexner type VI	—	Opalescence (mild).
Flexner type Y	1,280	Opalescence (heavy).
Boyd type II	1,280	—
Boyd type P274	1,280	Opalescence (mild).

removed and used in precipitin tests for identifying their type of Flexner organism. The nature of the active principle in the extract—whether it is a complete antigen or only a hapten—is not known. If the antisera are diluted as suggested, the reactions are in most cases confined to the type antisera.

The reactions of Flexner type IV are interesting. This is the organism, described by Boyd[7] under the name of type 103, which undergoes the variation 103A to 103B. Type 103A contains the type-specific antigen and is not easily agglutinated by the other Flexner type antisera. If this organism loses the type-specific antigen and undergoes the variation to 103B, it is then agglutinated by all the Flexner type antisera. The standard type organism used in this investigation was agglutinated by all the Flexner type antisera, and it is possible that this variation had taken place, in which case the widespread reactions in the precipitin test can be understood. This activity may be due to the removal of the group antigen and the reaction of this with group antibody in the type antisera, or it may be due to the removal of the different type antigens which react with their own antisera as suggested by the Andrewes and Inman hypothesis. The problem awaits investigation. Whether a more specific reaction would be given by a freshly isolated strain can be determined only by trial in an area where this type is prevalent.

The value of the precipitin test is demonstrated also in the differentiation of Boyd type P274 and *Bacterium alkalescens*. The latter organism is agglutinated to high titre by the antiserum of P274, but in the precipitin test the same antiserum gave an obvious precipitate with Boyd type P274 and only a mild opalescence with *Bacterium alkalescens*. When the types which are late fermenters of lactose (Sonne type and *Bacterium dispar*) were tested, the Sonne type reacted only with its own antiserum in the precipitin test, whereas *Bacterium dispar* gave no reaction with any of the antisera tried.

The results of the precipitin test applied to locally isolated strains confirmed the findings obtained with the standard type cultures, and a sufficient number of the Flexner type II and Sonne strains were available for the results to be regarded as consistent for these types.

The agglutination test with the standard type cultures produced the usual cross-agglutination amongst the Flexner types and demonstrated the predominance of the different antigens in the different types. It also revealed that Flexner types I, II and V were agglutinated to a significant degree by the Salmonella antisera except *Bacterium paratyphosum B*. The other Flexner types and the Boyd types I, II and III were not agglutinated by any of the Salmonella antisera. The Sonne organism, on the other hand, was agglutinated by *Bacterium paratyphosum B* antiserum, but not by the other Salmonella antisera.

The Boyd types I, II and III organisms were not agglutinated to any extent by the Flexner antisera. The reactions of the antiserum of Boyd type I showed that some relationship exists between this organism, *Bacterium alkalescens* and the Schmitz type. Boyd type II antiserum agglutinated several of the Flexner type organisms; this suggests a sharing of antigens. *Bacterium dispar*, and particularly *Bacterium alkalescens*, were also found to share antigens with many other types. These agglutination reactions demonstrated how the dysentery bacilli are interrelated antigenically, not only with one another, but with some of the Salmonella organisms and with non-dysentery bacilli like *Bacterium alkalescens* and *Bacterium dispar*.

The agglutination reactions of the locally isolated strains resembled the agglutination "picture" of one of the standard type organisms. The reactions showed that, if one knew the standard reactions with antisera of the dysentery and Salmonella organisms, it was possible to place the unknown strains in their proper types. The results of the agglutination test were not so clear-cut as those of the precipitin test.

Under the conditions of the experiment, the Flexner types III, IV and V, the Schmitz type, *Bacterium alkalescens* and *Bacterium dispar* produced abundant indole. The locally isolated strains gave the same reactions as the standard types to which they belonged, with the exception of the two strains of Flexner type IV. These organisms failed to yield a reaction and thus differed from the standard type reaction. For this reason the indole reaction of this type must be regarded as variable. When only standard types were available, the results of the test for indole must be tentative until enough strains of each have been examined. This applies mainly to the Flexner and Boyd types, because the results of the test with the Shiga and Schmitz types, *Bacterium alkalescens* and *Bacterium dispar* were the same as those published in the text-books.

The reactions to the methyl red test have been given; but the locally isolated strains varied in their reactions in some cases from those of the standard type cultures, and the test was of no value for identification purposes.

There are three organisms—Flexner types X and Y and Boyd type P274—whose classification is considered doubtful. This investigation has furnished some evidence which might aid in their classification, and for this reason they will be discussed separately.

According to Boyd,[3] Flexner type X is an incomplete variant of Flexner type III, and Boyd states that Andrewes's strains of type X have now entirely different characters from those that he (Andrewes) described. In the present work the reactions of Flexner types X, III and II showed how closely the last-mentioned organism was related to the X type. The precipitin and indole reactions were the same and differed greatly from those of Flexner type III. The type X organism was agglutinated to full titre by type II antiserum, but only to one-eighth of that of type III. Both type II and type X were agglutinated by the Salmonella antisera, whereas type III gave no such reaction. These reactions showed that the strain of type X used in this investigation had properties which allied it rather to Flexner type II than to Flexner type III.

There are various types of the Y organism. Perhaps the two most discussed are the Lentz type and the Hiss-Russell type. The former has been identified by Boyd[3] with his type 103 or type IV under the new nomenclature. He suggests that the Hiss-Russell Y is a group variant of type II. In the present work two Y strains were available. These were the standard type culture of Hiss-Russell Y, and the strain obtained from the University of Adelaide. The reactions and history of the latter organism have been given; but whether it was a Lentz or Hiss-Russell type is not known. In this series certain of the reactions of Flexner type II were found to be constant. They were, firstly, that the extract from the organism reacted only with its own antiserum in the precipitin test; secondly, that it was agglutinated by the Salmonella antisera, and thirdly, that the test for indole produced negative results. The Flexner type IV organisms also showed some constant features. In the precipitin tests the extracts gave reactions with all the Flexner type antisera, and in the agglutination tests none of the organisms were agglutinated by the Salmonella antisera. The response to the test for indole was, however, variable. The two Y strains were similar, in that their precipitin reactions were the same as those of type IV. Like this organism, neither of the Y strains was agglutinated by the Salmonella antisera.

The strains differed from one another in their indole production. Taken as a whole, the reactions of the Hiss-Russell Y organism favoured its classification with Flexner type IV rather than with Flexner type II.

The work of Rothstadt, Fenner and Baker[8] is of importance because it shows that the organism known as Boyd type P274 exists in Australia. The work also demonstrates the pathogenicity of the organism as a cause of dysentery, a fact which was confirmed by Boyd in a personal communication to the authors. In the present investigation the organism was not agglutinated to any great extent by the Flexner antisera, nor did the precipitin test reveal any relationship with this group. On the other hand, the reactions of the antiserum of this organism showed a striking resemblance to those of Flexner type V antiserum. The test for indole produced no reaction, whereas all the type V organisms yielded positive reactions. The fact that the Boyd type P274 organism was agglutinated by the Salmonella antisera—a property limited mainly to certain Flexner types among the dysentery bacilli—makes its classification doubtful. Before this organism can be regarded either as a Flexner or as a Boyd type, many more strains must be examined.

SUMMARY.

1. The precipitin test is a simple method of typing the Flexner group of dysentery bacilli. The test can be used for differentiating between Boyd type P274 and *Bacterium alkalescens* and between the Sonne type and *Bacterium dispar*. The reactions of locally isolated strains agreed with those of the standard type cultures.

2. The agglutination reactions demonstrated the extent of the sharing of antigens by the dysentery bacilli. They showed that the organisms of Flexner types I, II and V and of Boyd type P274 were agglutinated by the antisera of *Bacterium paratyphosum A* and *C* and of *Bacterium typhosum*, but not by that of *Bacterium paratyphosum B*. The Sonne type organism is agglutinated by *Bacterium paratyphosum B* antiserum, but not by the other Salmonella antisera to any great extent.

3. The results of the tests for indole were studied, and in tests with locally isolated strains were found to be consistently negative for Flexner type II and the Sonne type, positive for Flexner type V and variable for Flexner type IV.

4. The response to the methyl red test was variable for the Flexner types, and was found to be valueless for identification purposes.

5. The reactions obtained with Flexner types X and Y and Boyd type P274 indicated that type X is related to type II, that Hiss-Russell Y type is related to Flexner type IV, and that the classification of Boyd type P274 is uncertain.

6. Of locally isolated strains, 23 were Flexner type II; two (both from the same patient) were Flexner type IV; two were Flexner type V; and 14 were the Sonne type. The Sonne strains were isolated during an intrahospital infection, and the incidence of this type cannot be gauged from the figures. The Flexner types were isolated from dysentery patients who had been admitted to this hospital with that diagnosis.

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Reports of Cases.

NEPHROTIC TYPE OF NEPHRITIS IN ASSOCIATION WITH QUARTAN MALARIA.

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AND
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Clinical Record.

A FEMALE native child, approximately five years of age, was admitted to a Royal Australian Air Force hospital on a south-west Pacific island on October 27, 1943. A vague history was elicited of intermittent fever present for several months and swelling of the abdomen and legs of one week's duration.

The child was pale and ill and considerable œdema of the legs, vulva and lower part of the back was present. The face and eyelids were puffy and the abdomen was tense with ascites. The spleen was firm, enlarged, and palpable by dipping two inches below the costal margin. There were signs suggestive of a small pleural effusion at the base of each lung. The systolic blood pressure was 98 millimetres of mercury and the diastolic pressure was 60 millimetres. The tendon reflexes were brisk.

The urine was scanty in quantity, and when it was boiled three-quarters of its volume was albumin. Microscopic examination of the centrifuged deposit revealed numerous granular, cellular and hyaline casts and a few red blood cells. Examination of a thin blood film taken on the day of the child's admission to hospital showed the presence of malarial parasites—small schizonts containing eight to ten merozoites in a normal sized red cell, without Schüffner's dots. Examination of a further film on the next day revealed typical band forms of *Plasmodium malariæ*, together with trophozoites and gametocytes of *Plasmodium vivax*. The hæmoglobin content of the blood was 7·5 grammes per 100 cubic centimetres. Numerous hookworm ova were found in the fæces.

The patient was treated with quinine bisulphate, 0·5 gramme per day, for three days, then "Atebrin", 0·15 gramme per day, for five days, and finally "Plasmoquine", 0·01 gramme per day, for five days. Three grains of ferrous sulphate were given three times a day during the stay in hospital. The diet was unrestricted, but consisted mostly of carbohydrate. The hookworm infection was treated with carbon tetrachloride after the more acute symptoms had subsided. A secondary yaws eruption developed, which yielded rapidly to "Mapharsen", and she contracted influenzal pneumonia, which responded to sulphathiazole.

In spite of these varied complications the nephritic manifestations diminished slowly but steadily. On December 21 no œdema of the legs could be detected, nor did there appear to be fluid in the peritoneal cavity. The spleen was just palpable. Examination of the urine revealed a white cloud of albumin; microscopic examination revealed only a few hyaline and granular casts and occasional red blood cells. The hæmoglobin content of the blood was 13·2 grammes per 100 cubic centimetres. No malarial parasites were seen in the thick blood film.

Discussion.

A nephrotic type of nephritis, said to be due to infection with *Plasmodium malariæ*, has been reported from several localities where quartan malaria is prevalent. Unfortunately no references are available here; but it is our impression that no such cases have been recorded in the literature from the south-west Pacific area. The causal connexion between the malaria and the nephritis is difficult to prove in this case. However, in view of the well-known association of the two conditions in other parts of the world and the satisfactory response to treatment, it is considered that their combination in this patient was significant.

Acknowledgement.

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The Medical Journal of Australia

SATURDAY, JULY 22, 1944.

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PERTUSSIS: SOME ASPECTS OF ITS PREVENTION AND DIAGNOSIS.

MORE than three and a half years have elapsed since the control of pertussis was discussed in these columns. The object of the discussion on that occasion was to urge the use of Phase I vaccine as affording a valuable, though not complete, protection against the disease. In the same issue there appeared a paper on some aspects of pertussis immunization by Dorothy Gepp, who had been acting as medical research officer of the Children's Hospital, Melbourne. Since that time a discussion on pertussis immunization has been held by the New South Wales Branch of the British Medical Association, and an account of the meeting was published in this journal on August 30, 1941. In the New South Wales discussion the chief contributions were made by D. G. Vickery and Phyllis Anderson, and stress was again laid on the value of Phase I vaccine. Vickery in the course of his remarks referred to some of the difficulties met with in the matter, especially in private practice in this country. He pointed out that the Australian has a natural antipathy towards injections of any sort, especially if they are to be given to his children; that the Australian likes to be given 100% guarantee of protection; that he takes a great deal of rousing before he will adopt any medical procedure, unless he is faced with the disease in his midst; that he is strongly suspicious of reactions after injections. For these reasons Vickery thought that it might take a number of years before the public was persuaded to have children protected as a routine procedure. In his contribution to the discussion, Professor H. K. Ward raised the question of how many of the susceptible would have to be immunized in order to make a dent in the mortality figures. The whole question, he insisted, depended on the effective inoculation rate. This had been fixed by Ledingham at 80% of susceptible individuals; but Professor Ward regarded that figure as rather pessimistic and thought that 60% might be the figure. At the same time he said that if the State was persuaded to take on the task of immunization against pertussis, it would be taking on something more difficult than immunization against diphtheria. The American experiments, he said, had shown that with a certain technique it was possible to establish a worthwhile immunity against pertussis. If the technique used in the American experiments was modified to facilitate mass immunization, it would be necessary to carry out a field experiment to prove the value of the modified technique. This, Professor Ward stated, meant a good deal of hard work; there was no short cut.

The present intention is to bring to the notice of practitioners some recent happenings which, though perhaps belated, show that a desire to achieve immunization against pertussis is spreading. Before this is done the opportunity may be taken to mention a recent and important contribution to the diagnosis of the disease. This digression may be excused by the thought that if pertussis is not prevented, its occurrence must be discovered and the sooner the diagnosis is made the better. Robert Cruickshank, who is engaged in the pathological service of the London County Council, has reported a method of bacteriological diagnosis which he thinks could be readily adopted by the practitioner in the diagnosis of pertussis.[1] In this method a swab is introduced through the mouth and a swabbing is taken from the naso-pharynx; it is then inoculated on a "penicillin plate" of Bordet-Gengou medium. The value of the penicillin plate depends on the selective resistance of *Hæmophilus pertussis* to penicillin. Cruickshank found that inoculation onto the Bordet-Gengou medium was not particularly successful with either laryngeal or pharyngeal swabs when compared with the use of a cough plate. When penicillin is added to the medium the growth of other organisms is inhibited and *Hæmophilus pertussis* is easily recognized. The use of a swabbing from the naso-pharynx is held to be advantageous because it is particularly suited to suspected infections occurring in children seen at home or in the out-patient department. For details of this method and of the results obtained by Cruickshank with it the original paper must be consulted. It is a method about which more will certainly be heard in the future.

In regard to prophylaxis the value of immunization with Phase I vaccine has not been accepted as widely or as rapidly as it should have been. Apparently there has been no advance beyond the stage at which the value is stated and restated as though there are many unbelievers. An example will be found in our own review of J. H. Lapin's recent book on pertussis, published last December, in which the two statements quoted *verbatim* from the author dealt with the proof that has been given of the efficacy of pertussis vaccines in prophylaxis and with the availability of vaccine for use on a large scale under controlled conditions. How different are the statements made in regard to immunization against diphtheria. Underlying the pertussis position is the fact that, as Vickery put it, no 100% guarantee of protection can be given. From statements made from time to time the impression has been created that at least one of the State health departments would give this as a reason for its unwillingness to be wholeheartedly behind any attempt to push the use of Phase I vaccine as an immunizing agent. Be that as it may, the protection rate of 80% or more that has been reported among certain groups of children is being looked on as worth while by an increasing number of

[1] *The Lancet*, February 5, 1944.

people. An evidence of this was given not so long ago when one of the municipalities near Sydney approached the New South Wales Branch of the British Medical Association with a request for information on which action might be taken. This was mentioned at the recent meeting of the Federal Council. The matter was brought before the latter body because the New South Wales Branch Council had in mind the possible combination of immunization against diphtheria and against pertussis. The suggestion was made that the Federal Council might approach the Commonwealth Serum Laboratories to ascertain whether a combined immunizing agent could be prepared. The Federal Council, however, had information that the matter was being actively investigated at the laboratories.

Observations have been made in other places on the simultaneous administration to young children of diphtheria toxoid and pertussis vaccine. L. W. Sauer and W. H. Tucker reported in 1942[1] that since 1938 at the health department immunization clinic, Evanston, Illinois, 464 infant welfare clinic children, aged from eight to twenty-four months, had been given injections of the two antigens simultaneously. In order to determine whether the mixing of diphtheria toxoid and pertussis vaccine might lessen or augment the antigenic value of either or both components, the children were divided into two groups. One group consisted of 208 children who were given at three week intervals simultaneous injections of vaccine and, in the same arm, of diphtheria toxoid. The second group comprised 256 children who were given injections of mixed antigen, so prepared that each of the three doses given equalled the dose given in the other set of three simultaneous doses. By means of Schick tests, pertussis complement fixation tests and estimations of the concentration of diphtheria antitoxin in the serum, it was determined that the immunization responses to the mixed antigens compared favourably with those obtained after the separate injections. Work such as that of Sauer and Tucker shows that the two antigens can be combined, but further observations will have to be made before the effect of the combined use on a wide scale can be determined. If the combined immunization is to be acceptable to the public, the number of injections will have to be small and reactions negligible. For a section of the medical profession, including possibly some of the public health authorities, it appears that, unless ideas undergo a good deal of change, the success in diphtheria immunization will have to "sell" the pertussis immunization. But there is always hope, not only that the understanding and appreciation of the public will grow, leading its members to grasp proven opportunities though they may not be invariably effective, but that our knowledge of immunology will grow and the improvement of our weapons keep pace with the growth.

Current Comment.

FOSTERING FOOD FADS.

No doubt the strong element of volition and the extended choice of viands have led to the association of nutrition with more fads and with more charlatanism than any other physiological necessity. We have ceased to be fussy about the sort of atmosphere we breathe, though our grandparents dreaded the night air and shut it out with curtain and closed door and window. Then there arose in the latter part of the eighteenth century a vogue of inhaling various gases. James Watt, the great inventor, stuck his head into the flue of one of his furnaces and felt very sick as a consequence, no doubt through carbon monoxide poisoning. By a fallacy known to logicians as the undistributed middle, he came to the conclusion that flue gases had high medicinal properties—all medicines are nasty, flue gases are nasty, therefore they are medicinal. Then in 1798 Dr. Thomas Beddoes established the Medical Pneumatic Institution at Bristol, where the breathing of various "airs" and vapours was systematically studied. We may forgive the large amount of hocus-pocus in this establishment, for it gave to the world Sir Humphry Davy and the anæsthetic, nitrous oxide. At present the only respiratory predilections are associated with altitude, and many medical men, not without good grounds, believe that there is an optimum elevation for certain human types, though the vast majority of us are compelled to live not far from sea level. But when we come to food we find an extensive hunting ground for the uncritical enthusiast and the crank, not to speak of the quack and the impostor. The number of medical men who take up a food fad is, as we might expect, small, but those who develop the complaint are often, strange to say, as crotchety and pedantic as laymen similarly affected. In a recent issue of the *Annals of Internal Medicine* there is an article by a doctor of medicine and Fellow of the American College of Physicians entitled "The Raw Food Diet: A Therapeutic Agent".[2] Nephritis, tuberculosis, various œdemas, Ménière's disease, migraine, urticaria, hypoglycæmia and obesity can all be alleviated or cured by restriction of the diet to raw vegetable substances. Apparently the rawness has a lot to do with the therapeutic potency, for, as the author ingenuously states, "the principal difference between the strict vegetarian and the raw food diets is that the former admits considerable cooking". After contemplating the title the reader might well expect an extended disquisition on the wealth of vitamins in raw fruits and vegetables and the devastating effects of cooking on these; but, no, there is little or no mention of vitamins. Nor does roughage come into the picture, for apparently the commanding virtue of raw vegetables is that they contain abundant potassium and little sodium, and it is just this ratio which confers such singular powers. Just how this ratio is disturbed by the baleful action of cooking is not made clear. A list of foodstuffs is given with the suggestion that the practising physician will find here all that is necessary. It is nevertheless reassuring to find in the succinct "Conclusion" at the close of the article: "The raw food diet is not suitable for extended use, nor for healthy individuals, because of certain inherent deficiencies."

In *The American Journal of the Medical Sciences*, published in February of this year, there is an article on honey and cow's milk as an exclusive diet for adult human beings.[3] The authors are an entomologist, presumably an authority on bees, two agricultural biochemists, presumably authorities on cows, a dentist and a professor of preventive medicine. The deficiencies of cow's milk as a diet for adults are set out as low concentration of iron, copper, manganese and frequently iodine, a low calorie value in relation to bulk and a content of nicotinic acid and of vitamin *D* which appear to be below what is regarded as adequate. The reader learns that dark honey is 4·6 times richer in calories, 5·3 times richer in iodine, 100 times richer in manganese and 7·3 times richer in copper than cow's milk, and is therefore the ideal addition to make milk suitable for the exclusive consumption of the adult. With singular honesty the authors tell us that of the five human experimentees all got furred tongues from the

[1] *American Journal of Public Health*, April, 1942, page 385; quoted in "The 1942 Year Book of Pediatrics", page 123.

[2] A. A. Holbrook: "The Raw Food Diet: A Therapeutic Agent", *Annals of Internal Medicine*, March, 1944, page 512.

[3] M. H. Haydak, A. E. Vivino, J. J. Boehrer, O. Bjorndahl and L. S. Palmer: "A Clinical and Biochemical Study of Cow's Milk and Honey as an Essentially Exclusive Diet for Adult Humans", *The American Journal of the Medical Sciences*, February, 1944, page 209.

second day of the test onwards, one had four bouts of diarrhœa, another had scybalous stools, several had twitching of the eyelid, another suffered from several attacks of "cramps in the stomach", one went down to a common cold and another to virus pneumonia. Needless to say elaborate analyses of blood and urine were carried out. The grand conclusion of this research is in the single sentence: "The diet proved to be adequate to support life, but not to prevent deficiency symptoms entirely." Perhaps we are now able to understand the description of the promised home in Palestine by the spies sent out by Moses, "a land flowing with milk and honey"!

IMMUNIZATION IN THE UNITED STATES ARMY.

In a recent paper, A. P. Long, a lieutenant-colonel of the United States Army, has given a useful review of the Americans' policy towards immunization of their soldiers.[1] During the Great War regular immunization was used against only three diseases: smallpox, typhoid fever and paratyphoid fever. Today, active immunization is attempted as a routine measure against these diseases and against tetanus as well; in special circumstances vaccination is practised against yellow fever, typhus, cholera, plague, diphtheria and scarlet fever; and passive immunization is used in the prophylaxis of tetanus, gas gangrene, and, occasionally, of measles and mumps. Moreover, the army has an interest in current researches on influenza vaccine, dysentery vaccine and gas gangrene toxoid. The author has given details of the methods used in each type of immunization mentioned.

For smallpox vaccination, glycerinated calf dermo vaccine is used. The method recommended is that commonly known as the multiple pressure method, and dressings are not applied to the vaccinated area. Vaccination is repeated every three years and also after any known exposure to infection with smallpox.

The "T.A.B." vaccine contains in each cubic centimetre 1,000 million typhoid organisms and 250 million each of *Salmonella paratyphi* (paratyphoid A) and *Salmonella schottmülleri* (paratyphoid B). The initial vaccination consists of three subcutaneous injections given over a period of two to three weeks. The first dose is 0·5 cubic centimetre, and the second and third doses are each one cubic centimetre. Immunity is maintained by the injection of 0·5 cubic centimetre of the vaccine each year.

Tetanus toxoid is administered subcutaneously in three injections of one cubic centimetre, the interval between successive injections being three weeks. Various stimulating doses of one cubic centimetre are subsequently given: one, a year later; one, prior to departure for a theatre of war (if this occurs more than six months after a previous injection); and one after receipt of a wound or before operative manipulation of wounds. In the past, injections of tetanus toxoid were often followed by sensitivity reactions, but this has rarely occurred since certain peptones were removed from the culture medium in which the organisms for the vaccine are grown. The results of immunization against tetanus have been very good. Only nine men have contracted tetanus while serving with the American army in this war; seven of them had not been immunized; one of the remaining two had not received an emergency stimulating dose of toxoid; and the other showed certain features not typical of tetanus.

The yellow fever vaccine now being used is prepared from chick embryo cultures. It is dried and stored at freezing temperature. A single dose produces an immunity which lasts at least four years. This protection is given to troops who are stationed in, or are travelling through, areas where yellow fever is endemic. The immunization has been so successful that none of the troops has developed yellow fever; but it is now well known that at one stage the immunization itself was complicated by a serious outbreak of jaundice. This began in March, 1942, reached a peak in June, and subsided completely by September. The illness had the clinical features of catarrhal jaundice; the patients usually recovered completely in about six weeks, but there was a case mortality rate of 0·2%. There was strong evidence that an icterogenic agent was present in certain batches of the human serum which was added to the vaccine during its preparation. The vaccine used since that time has been prepared without human serum, and more than a year has elapsed without an outbreak of jaundice that could be attributed to the immunization.

The anti-typhus vaccine is injected in three doses of one cubic centimetre each, with intervals of seven to ten days between successive injections. A stimulating dose is given every four to six months. Only those troops who are in epidemic areas are immunized.

Cholera vaccination is required for all troops stationed in, or travelling through, Asia, and in special circumstances may be used elsewhere. The vaccine is made from two East Indian strains of high virulence and stability, and contains eight million organisms per cubic centimetre. The first dose is 0·5 cubic centimetre, and the second, which is given seven to ten days later, is one cubic centimetre. A further dose of one cubic centimetre is given every four to six months, or if there is a danger of infection.

[1] *American Journal of Public Health*, January, 1944.

HÆMORRHAGE IN SEVERE LIVER DISEASE.

The great interest awakened all over the world in the common infectious type of jaundice, that is, infective hepatitis, has directed attention again to the rare but exceedingly worrying problem of the jaundiced patient who suffers from hæmorrhage. Occasionally in the midst of an epidemic of mild jaundice cases are seen in which prolonged jaundice gives rise to doubt as to the possibility of the presence of a surgical lesion in the bile passages. There are also cases which follow the course of the subacute hepatic necrosis, and these are most frequently seen when the disease arises after the parenteral administration of some immunizing agent, such as yellow fever vaccine, or of human plasma. Sometimes, contrary to our fears, patients in the latter category may recover. The occurrence or threat of hæmorrhagic episodes in cases of these types is very worrying, and any surgeon who has to undertake an operative procedure in such cases carries a great load of responsibility.

The relationship between hypoprothrombinæmia and lack of vitamin *K* in these grave dislocations of liver function is now fairly well understood, particularly since the recent work on the hæmorrhagic diseases of the newborn. However, it is one thing to understand the mechanism, but it may be quite a different matter to be sure of our ability to handle this difficult problem of the control of bleeding associated with severe liver damage. R. E. Kinsey has made a contribution to this subject recently and recommends the use of transfusions of blood from donors who have received doses of vitamin *K* within the twenty-four hours preceding the transfusion.[1] He claims that this method is an advance on the direct administration of vitamin *K* to the patient followed by blood transfusion. Only five cases are reported in this series, these being cases of acute liver atrophy observed in 663 cases of epidemic jaundice in a military camp. The plasma prothrombin content of these patients was so reduced that severe spontaneous hæmorrhages occurred. Vitamin *K* was administered by mouth in both natural and prepared forms, and was also given by intramuscular injection. Only one of these patients recovered, and his case history suggests that his degree of liver damage was less than that of the other four, but the point that the author makes is that the bleeding was controlled satisfactorily by the above method, even though death ultimately took place from infection or hepatic failure.

Though no alteration in the prothrombin level was found in the plasma of the prepared donors after the administration of the vitamin *K*, Kinsey's account of his cases records definite improvement following the special transfusions. Further observations of this method would seem to be worth while.

[1] *Archives of Internal Medicine*, February, 1944.

Abstracts from Medical Literature.

DERMATOLOGY.

Cutaneous Autosensitization: Role of Staphylococci in Chronic Eczema of the Hands.

H. H. HOPKINS AND E. L. BURKY (*Archives of Dermatology and Syphilology*, February, 1944) discuss the "id" concept, and first quote Guth, who used this term to label an exanthem occurring in children suffering with kerion, a fungous infection of the scalp. Guth showed that his patients reacted positively to intracutaneous injections of extracts of trychophytes. Then followed a series of experiments establishing the relationship of certain other eruptions, notably the so-called dyshidrotic vesicular eruptions occurring on the hands, to other fungous infections. These were called dermatophytids. Peck in 1930 reported on 23 patients suffering from dermatophytosis on the feet and simultaneous dermatophytids on the hands and body. A new "id" made its appearance in 1934. Barker in England, and later Andrews, Birkman and Kelly in the United States, described a curious persistent eruption on the hands and feet, which has since come to be regarded at least clinically as a specific entity. Barker first called this disease "pustular psoriasis", but later accepted Andrews's designation of "pustular bacterid". The authors state that up to this point they have been concerned entirely with the concept of the sensitization of the skin by circulating antigenic or toxic substances elaborated in a distant focus of infection. The possibility of the sensitization of the skin by a toxin or antigen elaborated *in situ* should be considered. Such a mechanism might conceivably explain the manner of production of many unexplained conditions called eczema. The authors then quote Burky's experiments. The latter, working with rabbits, attempted to sensitize them to lens substance, an organ-specific substance. He found that cutaneous sensitivity to lens substance could be produced only by repeated and coincident injections of lens substance *plus* staphylococcus toxin. With this experimental evidence in mind, the authors considered it was likely that staphylococcus toxin might be liberated in the skin by the indolent growth of a low-grade toxin-forming organism, that this toxin might combine with epidermal keratin, presumably an organ-specific substance, or a product of keratin, and that a local sensitization phenomenon in the form of dermatitis might result. If this is true, then patients in whom such a process has occurred should react to staphylococcus toxin and also to keratin. It should also be possible to recover toxin-forming staphylococci from the lesions of the skin. In 1938 one of the authors, H. H. Hopkins, presented at the Atlantic Dermatological Conference in Baltimore a patient suffering from intractable eczema of the hands, from whose lesions a toxin-forming staphylococcus was repeatedly recovered. E. L. Burky suggested that in this case the eczematous lesions might be caused by local sensitivity to keratin or some other component of skin brought about by the synergistic effect of toxin liberated at the site of the lesions. Since then the authors have treated more than 50 patients suffering from acute, subacute or chronic dermatitis of the hands. The group contained no patients suffering from known contact dermatitis. *Staphylococcus aureus* was recovered from the cutaneous lesions of 40 patients. In 30 of the cultures the organism was hæmolytic. The authors treated the patients with intracutaneous injections of toxin, given twice a week for a minimum period of three months. Treatment is begun with toxin in dilutions of 1:10,000. Ten patients out of a total of 20 treated with staphylococcus toxin were cured.

Sulphonamide Compounds in Erysipelothrix Rhusiopathiæ Infections.

J. V. KLAUDER AND A. M. RULE (*Archives of Dermatology and Syphilology*, January, 1944) orally administered sulphanilamide, sulphapyridine, sulphathiazole and sulphadiazine to mice in doses of 0·2 gramme per kilogram of body weight. To some mice the compounds were administered twice a day for two days before inoculation with a virulent strain of *Erysipelothrix rhusiopathiæ* and twice a day thereafter for six additional days. To other mice initial treatment was given four hours after inoculation and then twice a day for six additional days; to other mice again, treatment was given for eight additional days. The survival of 12·5% of mice treated with sulphonamide compounds after inoculation shows a limited therapeutic effect of the compounds, which is slightly enhanced when they are employed in conjunction with subcurative injections of immune serum. The authors then discuss sulphonamides in the treatment of erysipeloid infection in human beings. The limited therapeutic value of the sulphonamide compounds against the infection in mice does not suggest that they will be efficient in treatment of the infection in human beings. In the authors' experience no striking results were obtained with sulphonamide compounds in the treatment of erysipeloid. In one of the authors' cases four grammes of sulphanilamide were given for ten consecutive days without effect on the erysipeloid. The authors quote other observers who treated erysipeloid with sulphonamides without avail, and they themselves consider that the sensitizing capabilities of sulphonamide compounds alone should restrain their use in the treatment of erysipeloid, which usually pursues a self-limited course. Antiserum remains the treatment of choice for patients with severe forms of erysipeloid and for patients with the septicæmic form of infection. Combined therapy with serum and a sulphonamide compound is indicated for patients whose infection is of long duration.

Dermatology in an Army Station Hospital.

H. W. WOOLHANDLER (*Archives of Dermatology and Syphilology*, February, 1944) states that a special military therapeutic problem is presented in eruptions of the feet. The importance of good feet to a soldier needs no elaboration, yet it must not be supposed that chronic eruptions of the feet necessarily require the recruit's rejection. Many positions are available in the army which do not require excessive use of the feet, such as those of cook, clerk and chauffeur. Of the diseases peculiar to the feet, fungous infections are undoubtedly the most common. The majority of these are seasonal, are not incapacitating and rarely require any special consideration. The treatment is not much different from that given in civil life, patients with the milder infections being treated as out-patients and the more severely infected being admitted to hospital. The eczematous eruptions of the feet rank next in importance. Limited service will often be required for men with this type of disease, and even then prolonged and frequent treatment will be necessary. Many will have to be discharged from the service, but it is difficult to describe just how severe an eruption must be before this is justifiable. Other eruptions which may necessitate discharge or limited duty include *acrodermatitis perstans*, *keratoses palmares et plantares* and pustular psoriasis. Hyperidrosis of the feet with an attendant eruption, practically always limited to the heels and sides, is rather commonly seen. Pillsbury, Sulzberger and Livingood have referred to this condition as "symmetrical erythema", but the author prefers to call it "hyperidrotic dermatitis". There is no difficulty in obtaining a history of hyperidrosis of years' standing from these men, but the eruption usually appears just after the prolonged marching required by the army. If such soldiers are permitted to remain on their feet, treatment will be uniformly unsuccessful. On the other hand, the majority of the eruptions can be healed within twenty-four to seventy-two hours if the patient is admitted to hospital. Control of the hyperidrosis can be attempted with recognized measures, emphasis being placed on the use of aluminium chloride. Success is not always to be expected, and soldiers having frequent recurrences will require reclassification.

Dermatitis due to Shoes.

C. SHAW (*Archives of Dermatology and Syphilology*, March, 1944) reports his observations in 22 cases of dermatitis due to shoes. In every instance the diagnosis was confirmed by positive reactions to patch tests with the materials from which the shoes were made. New methods and materials employed in the preparation and manufacture of shoes may be responsible in part for the apparent increased incidence of this eruption. The author believes that the diagnosis is frequently missed, since the first impression of a vesicular dermatitis of the feet is that the patient has dermatophytosis. The salient features of shoe dermatitis are the following: (i) the appearance of the eruption shortly after wearing new or freshly dyed shoes and its disappearance after changing the shoes; (ii) a positive result to patch tests with the suspected shoe leather (or lining materials); (iii) the failure to demonstrate fungi by microscopic examination or cultural methods and the lack of effect of the harmful action of fungicidal therapy; (iv) the relatively greater itching than in dermatophytosis —the severity of the itching is out of proportion to that of the eruption and may be present several days before the eruption appears; (v) the lack of involvement of the interspaces of the

toes; (vi) the sharply outlined border of the dermatitis; (vii) the predilection for the great toes and the dorsa of the toes and feet instead of for the little toes and plantar surfaces and sides of the feet as in dermatophytosis (the eruption of shoe-leather dermatitis is usually most marked on the dorsa of the great toes and becomes progressively less on the smaller toes and may be entirely absent on the little toes); (viii) the involvement of the ankles when high shoes are worn. Ten cases are reported in detail. The manufacture of shoes is a complicated process. In addition to the many possible sensitizing chemicals which are used in the processing of the leather itself, the lining canvas in shoes usually is impregnated with antimildew compounds. There are many such substances, any of which may be used in shoes. In none of the cases was associated dermatophytosis found. Once the diagnosis is established by positive responses to patch tests, the patient usually may obtain relief by changing the brand of shoes.

UROLOGY.

Premature Ejaculation.

B. SCHAPIRO (*The Journal of Urology*, September, 1943) states that premature ejaculation is neither purely somatic nor purely psychic in character. Only a small proportion of cases are due to local changes in the genital organs, such as hyperæsthesia of the *glans penis*, abnormally short frenum or pathological changes in the posterior part of the urethra or prostate gland. The majority of cases fall into two classes. The first comprises sexual hypotonus due to physical, mental or sexual exhaustion and often associated with erectile insufficiency. This type responds to tonics and sexual rest. Secondly, the sexual hypertonic class is due to high sexual tension and is frequently associated with erection on slight stimulation. In such cases the condition is aggravated by tonics and sexual rest, but benefit is conferred by sedatives. The prognosis is better in the latter class. Psychically most patients are of the same type, being subject to over-anxiety, emotional instability and hesitancy. The combination and reciprocal influence of the psychic and somatic factors make them unable to exercise control over the ejaculatory mechanism. Therapy must be directed to the total personality, the aim being to break down unsound habit patterns and build up normal ones. Technical training and reconditioning of the ejaculation reflex is indispensable. Psychotherapy must be supported by dietetic and medicinal measures which appreciably shorten the period of treatment.

Reimplantation of the Renal Pelvis.

S. F. WILHELM (*The Journal of Urology*, September, 1943) draws attention to the large proportion of unsuccessful plastic operations on the uretero-pelvic junction when the continuity of the ureter is broken either accidentally or surgically. To avoid the frequent post-operative obstruction the author proposes implanting the distal cut end of the renal pelvis into the ureter. The ureter is incised for one centimetre at its upper end to make two "straps". The conical apex of the pelvis is inserted into the ureter and the "straps" are attached to the external surface of the pelvis by four chromicized gut sutures. A nephrostomy drain and ureteric catheter splint are used, as in other plastic operations.

Injuries of the Ureter.

R. B. MCIVER (*The Journal of the American Medical Association*, April 15, 1944) states that wounds of the ureter alone are rarely encountered, either in civil or in military practice. Such injuries should be treated surgically, promptly, and with provision for adequate drainage. Ureterostomy, pyelostomy and nephrostomy are employed to combat infection and obstruction. In selected cases, ureterointestinal anastomosis may save the kidney, but nephrectomy is often necessary.

Renal Injuries.

A. J. SCHOLL (*The Journal of the American Medical Association*, April 15, 1944) states that most renal injuries are mild, causing only slight pain, hæmaturia and moderate abdominal rigidity. Many penetrating renal injuries require no treatment, and exploration is called for only when there is persistent or recurrent hæmaturia, or when free perirenal or intraperitoneal hæmorrhage is suspected. Excretion urography can be used early, and has proved of great value in many cases. If exploration is decided upon, it may most often with advantage be delayed till the patient recovers from primary shock. Drainage of the renal area is the most satisfactory early procedure, especially in the case of penetrating wounds. It is simple and brief, and permits evacuation of clots, removal of fragments of tissue and foreign bodies, and packing to control hæmorrhage. Nephrectomy is indicated when excessive hæmaturia is persistent, or when severe damage to the renal pedicle or massive pulping of the kidney has occurred.

Renal Trauma and Hypertension.

A CLINICAL study of 50 cases of renal trauma by W. F. Brasch and G. W. Strom (*The Journal of Urology*, November, 1943) indicates that, although it may be related to hypertension, renal injury is not a common cause. The value of conservative treatment in renal trauma is exemplified by the fact that nephrectomy was required in only five cases. Hypertension existed in three, but the blood pressure returned to normal after removal of the kidney. In six other cases moderate degrees of hypertension were present before the injury or were due to extrarenal causes. In only one case was renal trauma considered a possible factor in the production of hypertension. When secondary infection occurs, hypertension is more prone to develop.

Uretero-Intestinal Anastomosis.

C. G. HIGGINS (*The Journal of Urology*, December, 1943) advocates transplantation of the ureter into the recto-sigmoid for extrophy of the bladder before the child is one year old. The infant tolerates such surgical procedures well. Recurrent attacks of renal infection may cause death before the child attains four years. Organisms in the bowel of an infant are probably less virulent than those in older children. Ureteral dilatation due to uretero-vesical obstruction may render anastomosis technically impossible at a later stage. Meticulous care in pre-operative and post-operative treatment is imperative for success and the operation is technically more difficult in young infants. Post-operative complications have been of little significance in the 22 cases in which operation was performed. Two infants died in this series.

Carcinoma of the Prostate Gland.

P. J. HAHLE AND H. T. BEACHAM (*Urologic and Cutaneous Review*, January, 1944) state that carcinoma of the prostate has been, and in spite of recent promising developments still is, a disease about which there is not the slightest reason for complacency. Among the lower social strata of the community in New Orleans the outlook in connexion with this disease is depressing in the extreme. In a four-year period recently, 88 patients died out of 342 admitted to hospital suffering from carcinoma of the prostate—just over 25% mortality rate. Early diagnosis is made with difficulty, chiefly because the onset is insidious and the early clinical picture is obscure. As a result, patients are seldom seen until the disease is far advanced and the general condition is poor. The causes of death are various, and include, as well as the disease itself, urinary tract infection and degenerative diseases common to elderly men. Radical perineal prostatectomy is indicated only in the earliest cases—that is, in very few—and is attended with a high mortality. Perurethral resection is the most practical operation for general purposes. The recent development of the use of castration and of stilbœstrol therapy has permitted results not hitherto achieved by any other form of therapy. Neither method is curative, but both cause at least temporary improvement in the general condition, relief from pain, local regression of the primary neoplasm and regression of metastases. Adequate dosage of stilbœstrol achieves just as good results as castration, and is to be preferred to the latter. The drug should be administered in oil, by intramuscular injection. The authors question the efficiency of oral administration. Adequate therapy is achieved by not less than 40 daily injections of five milligrammes each, followed by the injection of five milligrammes three times a week until the prostate is clinically normal to palpation.

Blood Phenol in Renal Failure.

P. R. ROEN (*The Journal of Urology*, January, 1944) describes a new method of estimating the blood phenol content which utilizes the Millon reaction and the photo-electric colorimeter. This method has the advantage of being specific for phenol. By this method it is confirmed that the phenol level in the blood is increased in renal failure accompanied by uræmia, regardless of the type of renal failure. It is not elevated in simple blood nitrogen retention without uræmic symptoms. The height of the blood phenol level, though generally related to it, is not directly proportional to the intensity of the uræmic state. The blood phenol level plays an important role in the pathogenesis of the uræmic syndrome.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Section of Neurology, Psychiatry and Neurosurgery of the New South Wales Branch of the British Medical Association was held on June 15, 1944, at Broughton Hall Psychiatric Clinic. The meeting took the form of a series of clinical demonstrations by DR. S. EVAN JONES, the medical superintendent.

Chorea.

Chronic Sydenham's Chorea.

The first patient shown was a female, aged eighteen years, who had been admitted to hospital on April 11, 1944; her condition was a contrast to the chronic progressive types of chorea. The patient had suffered from pertussis, measles and rheumatic fever in childhood. Choreiform movements were first observed at the age of six years. Since that age she had spent most of her life in hospitals; after admission to hospital she usually made considerable improvement, but when she returned home she quickly relapsed. At about the age of fourteen years she recovered sufficiently to be able to take a position for some months.

On her admission to Broughton Hall, choreiform movements were in evidence in the left upper and lower extremities and in the left side of the face. Although she was able to walk, she did so clumsily, in a jerky fashion and with her feet plantar-flexed. With regard to her mental state, at first she was resentful and morose; but she made a good social adjustment to the hospital environment. The choreiform movements had disappeared.

Attention was drawn to the attitude of her left hand, which was flexed at the wrist and over-extended at the metacarpo-phalangeal joints. If one watched this left upper extremity when the patient was walking, one observed that it swung loosely and the hand "waggled" about in an almost flail-like manner; the appearances indicated a considerable degree of muscular hypotonus and suggested cerebellar dysfunction.

Dr. Evan Jones pointed out that although relapses were frequent enough in the course of Sydenham's chorea, it was not often that one saw a patient with a history of the disease of such long duration as in the case under discussion.

Huntington's Chorea.

The second patient shown was an unmarried man, aged thirty-four years, who had been admitted to hospital on March 8, 1944. It was stated that four years earlier it had been noticed that his feet were out of control and that he laughed without cause. He impressed his friends as being "silly". He was called up after the outbreak of war and served for six months in the Militia. Recently he had been dull and quiet and had become fatigued quickly.

On his admission to hospital, he was found to be irrelevant and unreliable in his statements; he carried out commands incorrectly and his memory for both recent and remote events was poor. He was lacking in affective tone, although his face exhibited many fleeting expressions. His speech had a jerky, explosive quality. He displayed almost continuous purposeless movements of body, limbs and face, and he frequently struck and maintained bizarre attitudes for an appreciable time.

It was recognized that his condition was one of mental deterioraton—dementia—but at first the movements were regarded as the stereotyped attitudinizing of a schizophrenic. Further observation, however, carried a conviction that they were choreic in character. Later investigation revealed that the patient's mother had died in a mental hospital, that she had exhibited choreiform movements, and that her mother had also died insane (it was a reasonable inference that the last-mentioned was choreic too).

Dr. Evan Jones pointed out that the patient was the third in direct line of descent to suffer from chorea; unfortunately no information was available concerning collateral lines. He had three brothers, but nothing was known about them. The movements of the patient affected the face (inducing mimetic facial "attitudes" resembling normal emotional expressions) and the trunk and limbs (in the latter case producing attitudes which often had a theatrical character).

Early Huntington's Chorea with Onset Simulating Hebephrenia.

The third patient shown was a female, aged twenty-three years, who had been admitted to the hospital on June 1, 1944. She was the daughter of a choreic mother, who died in a mental hospital at the age of fifty years. The patient's maternal grandfather, a maternal aunt, two maternal uncles and a sister were choreic. This girl was the youngest of seven children. Her school record and her early record after leaving school were good; but for three years it had been noticed that she had been solitary and had not "got on" with other members of the family. Twelve months earlier she had fallen from a horse and probably sustained cerebral concussion, and since then she had become more solitary and irritable. The patient complained of tiredness, weariness and inability to work. At times she had periods of blankness in which she might drop things. She attributed her symptoms to the accident.

On her admission to Broughton Hall, she was "inhibited", dull, emotionally unstable, vague and dreamy; she expressed ideas that people were against her. It was noticed that she was manneristic, and that she frowned and twisted her mouth.

Dr. Evan Jones pointed out that the patient had first been referred to the clinic as a schizophrenic, and she had been so accepted at first; in fact, she was not unlike the hebephrenic type of patient. However, occasionally one observed sudden abrupt movements, for example, of the hands, and when she was walking an appearance of clumsiness in turning; these were in all probability early choreic manifestations. The mental condition was actually one of early organic dementia.

Huntington's Chorea in Sisters.

A female patient had been admitted to Broughton Hall in 1937 at the age of thirty-two years. Her father had died of Huntington's chorea in another mental hospital at the age of seventy years; her paternal grandfather had died at the age of fifty years and was probably choreic. A paternal aunt and two paternal uncles were choreic. Her maternal grandmother had died in a mental hospital of senile dementia, and a maternal grand-uncle was insane at the age of twenty-one years. The history of the patient's illness was as follows. It was first observed that the patient was making restless, irregular movements in 1935; they progressively became more pronounced, and her voluntary movements became clumsy and unreliable. At the same time an intellectual change set in, first in lowered capacity for attention and concentration. She had been employed at clerical work, but had to give this up. She tired easily and had lost weight.

On her admission to hospital, she exhibited continuous choreiform movements which were especially noticeable in the face; her speech was mumbling and indistinct and she made frequent grunting noises. With regard to her mental state she exhibited slight dementia. She was discharged from hospital on June 11, 1937, and readmitted on May 16, 1944. At the time of the meeting she was aged thirty-nine years. It had been learnt that her sister, another aunt and a cousin were also choreic. It had been reported that the patient had great difficulty in walking and had had frequent falls; she had become quite helpless in regard to washing and feeding herself, and she had difficulty in swallowing and dribbled a lot. There was an appearance of dementia which was probably greater than the reality; she could still read, and her memory appeared to be good. She was practically confined to a chair life, but could walk with the assistance of two persons. While walking, her legs tended to cross in scissors fashion. Choreiform movements were constantly present in the face, limbs and trunk; there were short intervals in which a limb or the face would alone become contorted. The movements in the face were grotesque caricatures of expression, and frequently the mouth gaped widely and the lips were pursed. Her hands frequently assumed and maintained for a time a posture of flexion at the wrist suggestive of the canine begging attitude. Her speech was practically unintelligible and was interrupted by animal-like grunts.

The sister of the previous patient, aged forty-two years, had been admitted to hospital on May 16, 1944. Her school and occupational record were unsatisfactory; she would never remain long in any position, and was always "a bit of a tartar" at home. She had not worked since she was twenty-five years old. Her mother had noticed choreiform movements only for twelve months, but stated that the patient's gait had been peculiar for five or six years.

On her admission to hospital, her mental state was expressive of resentment and annoyance, and she had preserved this attitude since; she actively fought any attempt to examine her and was generally obstructive and non-cooperative. She exhibited generalized incoordinate irregular involuntary movements of the same type as those shown by her sister, but to a less degree.

Dr. Evan Jones remarked that these two patients represented the third generation of known choreics; the origin

of the family was Kent. In this particular strain the choreic element seemed to evolve fairly rapidly, and in several members had led to death at the age of about fifty years.

Chronic Progressive Chorea, Probably Huntington Type.

A male patient, aged forty-eight years, had been admitted to hospital on March 21, 1944, with a history that for eighteen months he had "gone in the knees", dragged his feet and fallen about. Since Christmas his voice had changed and he had had difficulty in swallowing. He had lost a great deal of weight. Previously he was bright, active and "a good mixer". Irregular, jerky movements of the hands and body had been observed. Mental deterioration had occurred.

On his admission to hospital, mental examination revealed gross retardation and poverty of thought; his replies to questions were often irrelevant and frequently no replies could be secured. He was dull and apathetic and manifested little emotional activity. Physical examination revealed that all movements on command were performed in an irregular, jerky manner and were overlaid with numerous accessory movements. His speech was throaty and "jerky". The patient's father had died at the age of sixty-five years of Bright's disease; his mother was living and healthy at the age of eighty. His paternal grandfather lived to eighty years and his paternal great-grandmother to ninety-nine years. No information was available about his paternal grandmother. His father's eldest brother died at the age of sixty years, and was reported to have had queer movements and difficulty in walking. That was the only positive evidence of antecedent chorea which appeared sufficiently definite to justify the conclusion that the chorea came through the father's family; the father had either died before developing chorea or had had mild symptoms which escaped the notice of the informant.

Dr. Evan Jones pointed out that the patient gave a typical representation of Huntington's chorea in the early stages; the age of onset was the usual one, and he showed parallel evolution of choreic movements and mental deterioration. It was interesting to note examples of longevity in this family. In Dr. Evan Jones's experience long lives were quite common in families affected by Huntington's chorea, but he was not in a position to say that they were more common than in non-choreic families.

Transmission of Huntington's Chorea.

Dr. Evan Jones then discussed the transmission of Huntington's chorea. He said that it behaved as a Mendelian dominant character. If *C* represented the possession of chorea-carrying elements and *O* the absence of choreic elements, a pure-bred choreic would be represented by *CC*, a hybrid by *CO* and a normal person by *OO*; both *CC* and *CO* persons would manifest choreic symptoms. The matings usually occurring in choreic families would be of a chorea-tainted person (a hybrid choreic) and a normal person, for example: *CO* = *OO*. This would give two *CO* and two *OO* persons; in other words, 50% of the progeny would be hybrid choreics and would manifest choreic symptoms, and 50% would be normal and incapable of transmitting chorea.

Under certain conditions—for example, if a choreic family was living in an isolated community—the mating might occur of two tainted (hybrid) choreics, for example: *CO* = *CO*. This would give one *CC* person, two *CO* persons and one *OO* person; in other words, 25% of the progeny would be pure-bred choreics, 50% would be hybrid choreics and 25% would be normal; 75%, therefore, would manifest chorea.

From such a mating as the latter would occasionally appear a pure-bred choreic, and no matter what his mating, all his progeny would manifest chorea. If he mated with a normal person, such a mating would give four hybrids *CO*, all of whom would manifest chorea. If he mated with a hybrid, the result would be two pure-bred choreics and two hybrids, all of whom would manifest chorea.

Senile Chorea.

A female patient, aged sixty-four years, unmarried, had been admitted to hospital on March 15, 1944. Her father had died of pernicious anæmia in old age and her mother of senile dementia. Two brothers and a sister were living and two sisters and a brother had died. There was no record of any member of the family suffering from chorea. The patient had worked for many years as a children's nurse, and was said to have been capable and to have made good social adjustments. For some months she had been hypochondriacal, and had always had some complaint; she feared that she was losing the use of her legs. She had not slept well and had become rather depressed. Her thyreoid gland had been removed four months earlier.

On her admission to hospital she exhibited some degree of emotional depression; her memory was unreliable, and she was evasive and lacking in normal interests. The picture was one of mild depressive psychosis with early dementia. Her systolic blood pressure was 130 millimetres of mercury and her diastolic blood pressure 80, and it was noted that her movements were "rapid and staccato"; but she was not at the time identified as choreic.

Dr. Evan Jones said that the patient's depressive symptoms had cleared up with electric shock therapy. Further observation had revealed that she had slight but definite choreiform movements; they consisted of shuffling or fidgety movements of the feet, hands and shoulders and were of small amplitude. In the absence of family history, the patient had to be regarded as suffering from senile chronic progressive chorea. The condition was at an early stage, and the choreiform movements were bracketed with a certain amount of dementia.

Congenital Dementia Paralytica: Rapid Reversal of Serological Findings.

A female patient had been admitted to Broughton Hall at the age of twenty-five years in March, 1942. She was the youngest of five children; her father, aged sixty-three years, was living and said to be healthy; her mother had died suddenly at the age of fifty-two years. The patient was reported to have had a good school record, and had subsequently been employed as a shop assistant for five or six years; she had worked until two weeks before her admission to hospital. She had complained for some time of pain in her stomach, and had had "queer turns" and vomiting. She had become suspicious and thought people were talking about her, tormenting her and stealing her tea and sugar.

On her admission to hospital, she expressed mild paranoid ideas; apart from this she exhibited a degree of mental defect that was inconsistent with a good school record. It was concluded, therefore, that the condition represented a dementia rather than a congenital deficiency. She was undersized and looked younger than twenty-five years; she had kyphosis and scoliosis. Her upper incisors were missing. The pupils were unequal and reacted sluggishly to light. The Wassermann test applied to the blood serum and cerebro-spinal fluid, the Kline test and the gold sol test, all produced positive results. There was no history of acquired syphilis, and although there was little indication of parental syphilis, a diagnosis of congenital *dementia paralytica* was made on the basis of the patient's age and on her physical stigmata. It was recognized, however, that she could be suffering from acquired *dementia paralytica*. She was inoculated with malarial blood and had five rigors. She was also given nine injections of three grammes each of tryparsamide between April and August, 1942. She was discharged from hospital in July, 1942.

She was readmitted to hospital in May, 1944. Her friends related that she was unemployable, dirty in habits and irritable and quarrelsome. She expressed ideas that members of her family were not treating her "right". She had *pediculi capitis* and was unkempt and dirty. Her pupils did not react to light. All tests for syphilis applied to the blood serum and the cerebro-spinal fluid produced negative results.

Dr. Evan Jones said that the patient was regarded as suffering from arrested congenital *dementia paralytica*. The reversal of serological findings within two years and after relatively little treatment was unusual. The social result was not satisfactory, but this had to be attributed to the stage at which treatment was instituted, dementia being already well advanced.

Amyotrophic Lateral Sclerosis: Paraplegia in Flexion.

A female patient, aged fifty-two years, had been admitted to hospital on February 2, 1944. According to her husband the family history was unimportant. The patient had come to Australia seven years earlier, and her illness commenced about that time, with weakness and unsteadiness in her legs; this gradually progressed, and for twelve months she had been unable to stand. Her hands also had become weak and unsteady. There had been no indication of mental weakness. She had had precipitate micturition.

The patient said that she first noticed weakness of the legs while getting out of a bath about ten years earlier. She could then walk well, but about five years later she had to use a stick. Her legs gradually grew weaker; the right leg was always worse than the left. She used to fall from unsteadiness and weakness, but did not stagger; her legs did not feel stiff and she could move them quite freely but weakly. Her condition had deteriorated gradually, and for twelve months she had been confined to a chair. Her legs had been "doubled up" for six months and were growing

worse in this respect. For two or three years she had been unable to hold her urine properly; she had normal sensation, but could not hold it for long. Her voice had become hoarse; she spoke with a hoarse intonation, but quite clearly and without tremor, and the pitch was rather high. She had noticed some weakness of both arms, worse in the left than in the right; sometimes the hands opened quickly and she dropped objects; her back felt weak. Her bowels were not constipated; sometimes she had precipitate defæcation. She had had no pain, tingling or numbness, no dizziness and no giddiness.

Neurological examination revealed that olfaction was good. Vision was good; the visual fields were normal and the disks were pale. There was some apparent weakness of the *levatores palpebrarum*, otherwise the oculomotor functions were intact. The pupils were small; the left was larger than the right, and their reactions to light were sluggish. The sensory and motor functions of the trigeminal nerves were intact. The face had a paretic appearance and the mouth opened more widely on the right side than on the left. Hearing was normal. There were no subjective vestibular symptoms, but some nystagmus was present on left lateral gaze. Taste sensibility was normal. Paresis of the right side of the palate was present and the uvula was drawn over to the left. The tongue protruded on the mid-line; it was not atrophied and did not fibrillate. The palatal reflexes were not elicited, but the pharyngeal reflexes were active and the other cranial reflexes were present. The speech had a hoarse, high-pitched quality, of which the patient was aware; there was no tremor or slurring. No disorder of sensation was found. The patient had no paræsthesiæ. She was unable to stand, and she could not sit up from a recumbent attitude without help. There was almost complete paralysis of both lower extremities, which were ordinarily fixed in an attitude of flexion at hips and knees. Pronounced hypertonus of the hamstrings was present. The patient could with difficulty extend the left leg; she could not voluntarily extend the right. There was great resistance to passive extension on both sides. When the patient was sitting up, the legs extended under the influence of gravity. Paresis of both upper extremities, and wasting of the intrinsic muscles of both hands and of the fingers were present, the third, fourth and fifth fingers on both sides tended to adopt an attitude of extension at the metacarpophalangeal joints and of flexion at the interphalangeal joints. Intention tremor was present on the left side. The knee jerk on the right side was not elicited owing to hypertonus of flexors; on the left it was elicited. The Achilles reflexes were elicited, that on the left being clonic in type. Tests for syphilis failed to produce a reaction with the blood serum and the cerebro-spinal fluid.

The patient could voluntarily extend the left leg into an attitude in which the great toe was dorsiflexed and the other toes were "fanned" and plantar-flexed—in other words, into a "frozen" Babinski reflex. By passive straightening of the great toe, by pinching of the *tendo Achillis* or by deep pressure on the calf muscles, the left leg was caused to return to its earlier "doubled up" attitude; the great toe then lost its tonic attitude of dorsiflexion. The right lower limb could not be extended passively or voluntarily, but it extended itself under the influence of gravity when the patient was seated in a chair, and also when she was asleep. The left leg could be passively straightened, but against strong resistance by the flexors. In its "doubled up" position the flexors were strongly contracted and the extensors less so; in the extended position both flexors and extensors were strongly contracted. When the limb was in the flexed attitude, the patellar reflex was difficult to elicit on account of strong contraction of the flexors. When the limb was in the extended attitude the patellar reflex was difficult to elicit on account of generalized hypertonus. On both sides at times the plantar reflexes were extensor in type; at times also Rossolimo, Bechterew and Chaddock types of response could be elicited.

Dr. Evan Jones said that the diagnosis in this case rested on the presence of signs of involvement of (i) the anterior horn cells and bulbar motor nuclei (as seen in the wasting of the intrinsic muscles of the hands, and palatal paralysis), and (ii) the pyramidal tracts, as revealed by the paralysis of the lower extremities with the abnormal reflexes characteristic of pyramidal affections. In paraplegia two attitudes occurred. (i) The first was one of extension; this occurred in pure pyramidal (lateral sclerosis) lesions, the attitude being determined by the influence of extrapyramidal tracts, which established the value of tonus in the different muscle groups. In the extensor attitude all muscle groups were hypertonic, but special emphasis was placed on the adductors of the thigh and the extensors of the leg and foot, the effect being a lengthening of the limb. (ii) The second attitude was one of flexion. This appeared when the spinal lesion was diffuse and involved the extrapyramidal as well as the pyramidal tracts. The attitude was determined by predominance of tonus in the hamstrings and by relative hypotonus of the quadriceps group. Attention was drawn to an apparent paradox in the case under discussion. Dorsiflexion of the hallux on plantar stimulation was regarded as part of Babinski's "defence reflex" or Marie's flexion reflex; in other words, when a stimulus was applied to the sole of the foot or to some other reflexogenic area, the limb shortened (flexed) and the hallux dorsiflexed. In the present patient, on the other hand, dorsiflexion of the hallux appeared as part of the attitude of extension, while when the leg was flexed the dorsiflexion of the hallux disappeared.

Acute Toxic Delirium.

A female patient, aged fifty-two years, had been admitted to a general hospital early in April, 1944, with a history that she had been well until three weeks before, when she contracted a cold which left her with severe pain in the right side of the back; this later spread to the left side and was not related to respiration. When admitted to hospital she had pain in the back "worse after meals", no vomiting and no eructation; she had a sense of fullness in the epigastrium. She had lost about ten pounds in weight in two or three weeks. Her systolic blood pressure was found to be 190 millimetres of mercury and her diastolic blood pressure 115. X-ray examination revealed no abnormality in the chest and pronounced spondylitis in the thoracic region of the spine and to less degree in the lumbar region. A barium meal examination gave negative results. There was no official record of her progress while in hospital, but her friends stated that she vomited frequently and was given sedatives freely. She was referred to Broughton Hall because of the development of active mental symptoms. When she arrived there on May 23, 1944, she was in a state of acute confusion. Although she was accessible for a short time, she soon passed into a state of stupor which continued for twenty-four hours; she could not be aroused from this stupor to respond to questions, but deep pressure over the calves and the upper part of the arms produced an immediate response indicative of pain. Her general condition was one of extreme debility and she was regarded as being practically moribund. The mental state was obviously one of delirium and the evidence of physical examination indicated that generalized acute peripheral neuritis was present. The syndrome was then recognized to be Korsakow's psychosis. No information was available concerning toxic factors, but later it was learnt that there was no history of alcoholism.

Treatment consisted of injections of thiamin hydrochloride, ten milligrammes twice a day, and ten units of insulin per day. The patient responded slowly at first, showing alternate periods of acute delirium and deep stupor; but during the week prior to the meeting she had improved rapidly in strength, intervals of lucidity had become more prolonged and the polyneuritis had greatly receded.

Dr. Evan Jones said that the lesion was illustrative of a course of events that sometimes developed if due attention was not paid to the need for adequate vitamins, especially vitamin B_1, when patients were under fairly heavy sedation, and especially so if they were not taking food or were vomiting. Under such conditions a B_1 avitaminosis could develop with dramatic rapidity, and if its premonitory signs were not recognized it might advance to a Korsakow psychosis.

Post-Graduate Work.

WEEK-END COURSE AT LISMORE, NEW SOUTH WALES.

THE New South Wales Post-Graduate Committee in Medicine announces that, in conjunction with the North-Eastern Medical Association, it will hold a week-end course at Lismore on Saturday, August 5, and Sunday, August 6, 1944. The programme is as follows:

Saturday, August 5, 1944.

At the Lismore Base Hospital.

2 p.m.—General meeting of the North-Eastern Medical Association.

4.15 p.m.—"Diagnosis and Treatment of Conditions about the Knee": Major G. A. Berkheimer, 118th General Hospital, United States Army.

At the Commercial Hotel, Lismore.

6.30 p.m.—Official dinner.

7.30 p.m.—"The Use of Penicillin in Medical Practice": Major F. T. Billings, 118th General Hospital, United States Army.

Sunday, August 6, 1944.

At the Lismore Base Hospital.

9.45 a.m.—"Common Diseases of Babies and their Management": Captain W. C. Stifler, 118th General Hospital, United States Army.

11.15 a.m.—"Shoulder Ailments in the Army": Major G. A. Berkheimer.

2.30 p.m.—"Peptic Ulcer and Pseudo Peptic Ulcer": Major F. T. Billings.

4.15 p.m.—"Important Aspects of the Nutrition and General Care of Infants and Children": Captain W. C. Stifler.

The fee for the course will be one guinea. There will be no charge for members of the defence forces. Those intending to be present are requested to notify Dr. G. J. Rawle, Honorary Secretary, the North-Eastern Medical Association, Lismore, as soon as possible, also to state whether it is their intention to be present at the dinner.

Correspondence.

AN APPEAL.

SIR: The Medical Benevolent Association of New South Wales will be grateful to members of the profession for donations of used X-ray films.

We have received an offer from the Transparent Sheet Manufacturing Company of North Sydney, Box 4091W, G.P.O., 'phone XA 2245, to purchase the films at 2s. 2d. per pound. The company's carrier will pick them up in the metropolitan area when notified. Country parcels should be forwarded by rail to St. Leonards Station, Sydney, c.o. Mr. R. Stather, the company's forwarding agent. My association will be most thankful if the donors will instruct the firm to make the cheques payable to the Medical Benevolent Association of New South Wales.

Yours, etc.,

WALTER F. BURFITT, Honorary Secretary.

Elizabeth Bay,
New South Wales,
June 28, 1944.

THE NERVOUS DISORDERS OF SERVICE PERSONNEL: AN URGENT PROBLEM.

SIR: A number of the members of the undermentioned association have read with considerable interest your editorial on nervous disorders of service personnel in the issue of THE MEDICAL JOURNAL OF AUSTRALIA of June 3, 1944, and it is considered that these lay comments may prove of some interest to your readers.

Reference is made in particular to the observations contained in the latter part of the third column of the editorial where it is stated:

> This treatment should be carried out in special hospitals where the patients would be resident and not outdoor patients, and the resident staffs of these hospitals should include a sufficient number of trained psychiatrists. Secondly, hospitals of this kind should be in close liaison with an interested and capable employment agency. If such an organization could be brought into being and used with enthusiasm and wisdom, it might be possible to create something that is almost as essential as the organization itself—a new mental outlook, for patients and public alike, that will expect recovery instead of compensation.

To this association, it seems that "Rockingham" Red Cross Convalescent Home, which is situated in Barkers Road, Kew, in Melbourne, has already successfully put into practice the recommendations which you have made.

In this home, through which, during the last 34 months, over 700 non-commissioned personnel of every type have passed, the patients are resident and are under the care of trained psychiatrists who have, we can affirm, cared for us in every way possible, and have worked hard and successfully to secure congenial employment for those patients who have been in need of assistance in that direction on being discharged from the home and the services.

Perhaps special stress should be laid on the concluding sentence of the section quoted:

> If such an organization could be brought into being and used with enthusiasm and wisdom, it might be possible to create something that is almost as essential as the organization itself—a new mental outlook, for patients and public alike, that will expect recovery instead of compensation.

In the lay opinion of this association the wish expressed in your editorial has already been fulfilled, as we, being ex-patients, can vouch that, owing to "Rockingham", we have been transformed from "nerve cases" to men capable of performing and in fact creditably performing, we trust, our duties and tasks as good and useful members of society.

It is considered that the fact that this ex-patients' association has been formed, the membership of which now exceeds 250, whose purpose it is, in appreciation of what has been done for us as patients, to do whatever is possible to assist the work that is being done in this home, will provide final proof of the excellence of the methods employed in this home and of the thorough efficacy of the work which has been so splendidly and enthusiastically done by the psychiatrists and the staff.

Yours, etc.,

T. W. BOWERS, Vice-President.
H. BEAUMONT, Honorary Secretary.

Rockingham Ex-Patients Association,
19, Barkers Road,
Kew, Melbourne.
June 14, 1944.

AN ACKNOWLEDGEMENT.

SIR: The Council of the New South Wales Branch of the British Medical Association desires to express its sincere appreciation of the generous action of a medical officer of the United States Army, who desires to remain anonymous, in making a donation to the library of twenty pounds for the purchase of the following journals: *American Journal of Psychiatry, Psychiatry.*

Yours, etc.,

J. G. HUNTER,
Medical Secretary.

British Medical Association House,
135, Macquarie Street,
Sydney.
July 13, 1944.

A SEVERE SERUM REACTION.

SIR: Since I have been in practice it has been my good fortune not to encounter a severe serum reaction in any of my own patients, but a few nights ago I thought a strong man of thirty-five years was about to depart this life after receiving a hypodermic injection of one cubic centimetre of "A.T.S." (500 units). A small wound in his right hand had been sutured and then he was given the serum about 10.15 p.m. Outside the out-patient department of the hospital he spent some time and a good deal of energy in cranking up his car, and then drove as far as the corner of the street, when he felt so queer that he asked his friend to take over the wheel. This was his last recollection of subsequent events until he began to recover an hour later. When the car drew up at his front door he was carried to his bed as a stretcher case.

On my arrival at the house at 10.45 I found him in bed fully clothed and breathing deeply for more air. I felt a little reassured to note that his colour was good (I have since noted his naturally pasty complexion), but his pulse was quite imperceptible. He vomited undigested food, sweated profusely, had sibilant rhonchi at the lung bases, and his heart was beating feebly at the rate of 90 per minute.

At 11.15 his breathing became less laboured and his pulse was now perceptible. Before I left him at 11.45 I listened again to his chest and the rhonchi had completely disappeared. About 3 a.m. large patches of urticaria caused an irritation over most of his body surface and there was swelling of his lips and upper eyelids, which persisted until midday. He had completely recovered from his experience towards the end of the day. He had never before been given serum of any kind.

Yours, etc.,

K. S. MACARTHUR BROWN.

Parramatta,
New South Wales,
July 8, 1944.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned has been registered as a duly qualified medical practitioner:

Small, Ronald Herbert, M.B., B.S., 1923 (Univ. Melbourne), 32, Carlotta Avenue, Gordon.

The following additional qualification has been registered:

Procopis, George Sandford, Lidcombe State Hospital (M.B., 1936, Univ. Sydney), M.R.A.C.P., 1944.

TASMANIA.

THE undermentioned have been registered as duly qualified medical practitioners:

Pinkus, Numan, M.B., B.S., 1944 (Univ. Melbourne), Launceston General Hospital.

Rasmussen, Winifred Ruby, M.B., B.S., 1944 (Univ. Melbourne), Launceston General Hospital.

Gay, Keith Roy, M.B., B.S., 1936 (Univ. Sydney), D.T.M., 1938 (Univ. Sydney), Commonwealth Laboratory, Hobart.

Obituary.

WILLIAM ALGERNON ANDREWS.

WE regret to announce the death of Dr. William Algernon Andrews, which occurred on July 15, 1944, at Marrickville, New South Wales.

FELIX PAUL BARTLETT.

WE regret to announce the death of Dr. Felix Paul Bartlett, which occurred on July 8, 1944, at Typiton, St. John, Devon, England.

ARCHIE SHERIDAN COBURN.

WE regret to announce the death of Dr. Archie Sheridan Coburn, which occurred on July 16, 1944, at Mullumbimby, New South Wales.

Nominations and Elections.

THE undermentioned has applied for election as a member of the Tasmanian Branch of the British Medical Association:

Cebon, Leon, M.B., B.S., 1944 (Univ. Melbourne), Launceston General Hospital, Launceston.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Lippmann, Arthur, M.D., 1907 (Univ. Kiel), (registered in New South Wales, September 3, 1941, in terms of Section 17 (2) of the *Medical Practitioners Act*, 1938), 175, Macquarie Street, Sydney.

Puffett, Robert Delmont, M.B., B.S., 1939 (Univ. Sydney), "Delmont", Cliff Street, Milson's Point.

Woolnough, Esther Isla, M.B., B.S., 1944 (Univ. Sydney), 28, Boomerang Street, Haberfield.

Gunning, John Phillip, M.B., B.S., 1944 (Univ. Sydney), Newcastle Hospital, Newcastle.

Medical Appointments.

Dr. Alfred Leonard Cooper has been appointed Deputy Medical Superintendent, Department of Public Health, New South Wales.

Diary for the Month.

JULY 25.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 26.—Victorian Branch, B.M.A.: Council Meeting.
JULY 27.—New South Wales Branch, B.M.A.: Branch Meeting.
JULY 28.—Queensland Branch, B.M.A.: Council Meeting.
AUG. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 2.—Western Australian Branch, B.M.A.: Council Meeting.
AUG. 2.—Victorian Branch, B.M.A.: Branch Meeting.
AUG. 4.—Queensland Branch, B.M.A.: Branch Meeting.
AUG. 4.—Victorian Branch, B.M.A.: Legislative Subcommittee.
AUG. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 8.—Tasmanian Branch: Branch Meeting.
AUG. 11.—Queensland Branch, B.M.A.: Council Meeting.
AUG. 11.—Victorian Branch, B.M.A.: Ethics Subcommittee.
AUG. 14.—Victorian Branch, B.M.A.: Hospital Subcommittee.
AUG. 14.—Victorian Branch, B.M.A.: Finance Subcommittee.
AUG. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 15.—Victorian Branch, B.M.A.: Organization Subcommittee.
AUG. 16.—Western Australian Branch, B.M.A.: Branch Meeting.
AUG. 17.—Victorian Branch, B.M.A.: Executive Meeting.
AUG. 22.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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